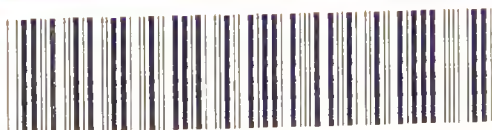


7/8


M18447





22101456143

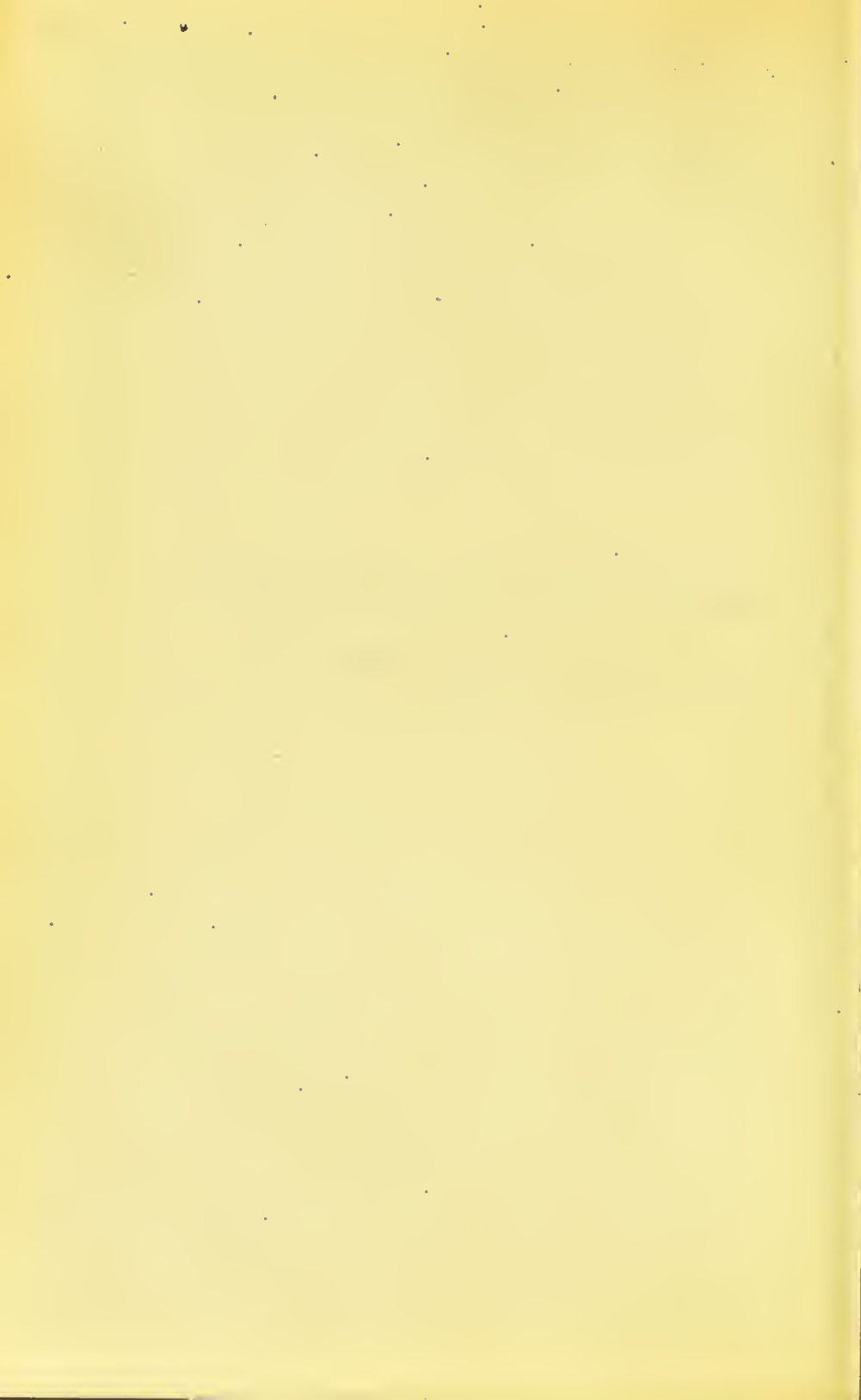


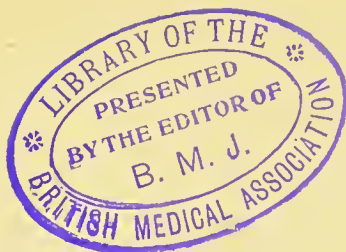


Digitized by the Internet Archive  
in 2014

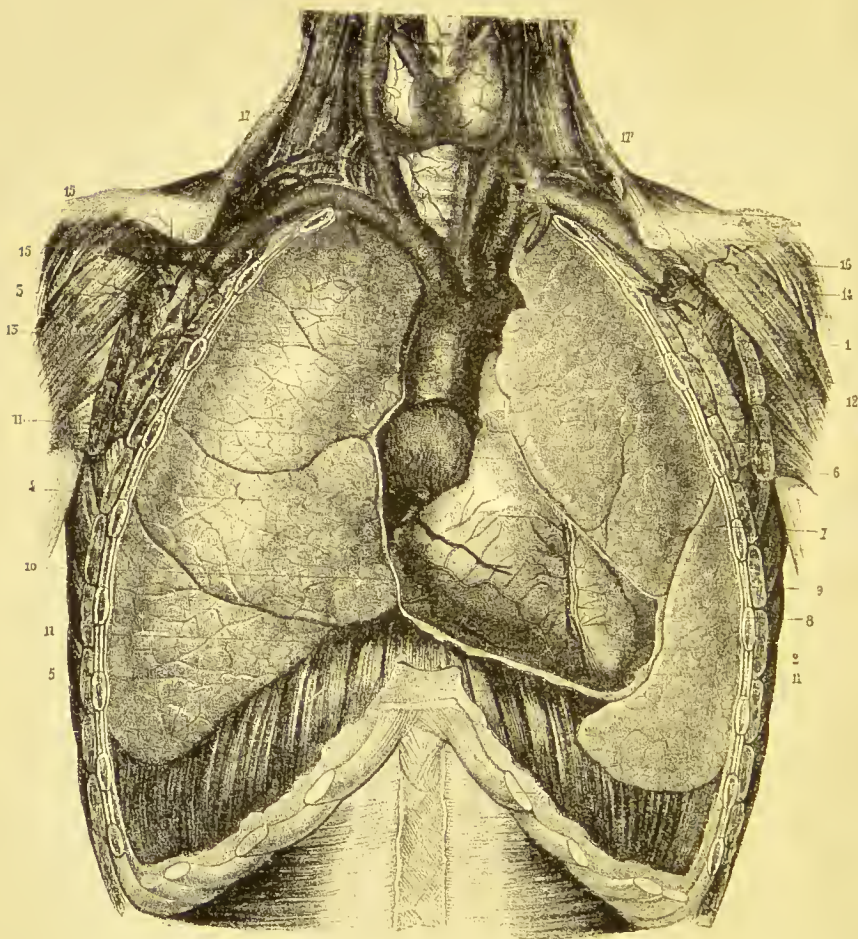


EXPLORATION OF THE CHEST  
IN HEALTH AND DISEASE





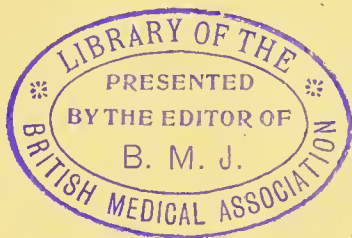




HEART, LUNGS, AND ARTERIES OF THE THORACIC CAVITY  
(BONAMY AND BEAU).

1, 2, left lung, divided into two lobes ; 3, 4, 5, right lung, divided into three lobes ; 6, right auricle ; 7, right ventricle ; 8, left ventricle ; 9, anterior notch ; 10, distribution of vessels upon the right ventricle ; 11, pericardium ; 12, pulmonary artery ; 13, aorta ; 14, innominate artery ; 15, right subclavian artery ; 16, left subclavian artery ; 17, right common carotid artery ; 17', left common carotid artery.

# EXPLORATION OF THE CHEST IN HEALTH AND DISEASE



BY

STEPHEN SMITH BURT, M. D.

PROFESSOR OF CLINICAL MEDICINE AND PHYSICAL DIAGNOSIS IN THE NEW  
YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL; PHYSICIAN  
TO THE OUT-DOOR DEPARTMENT (DISEASES OF THE  
HEART AND LUNGS), BELLEVUE HOSPITAL

LONDON  
H. K. LEWIS, 136 GOWER STREET  
1889

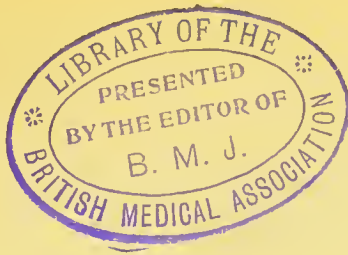
COPYRIGHT, 1889,  
By D. APPLETON AND COMPANY.

M18447

PRINTED IN AMERICA.

WELLCOME INSTITUTE LIBRARY	
Coll.	welMOMec
Call	
No.	WF970
	1889
	B97e





TO

D. B. ST. JOHN ROOSA, M. D., LL. D.,

IN APPRECIATION OF HIS FRIENDSHIP,

AND IN ADMIRATION FOR HIS MANY ATTAINMENTS,

THIS BOOK IS INSCRIBED

BY THE AUTHOR.



## P R E F A C E .

---

TEACHING naturally leads to requests from the class for a work that shall embody the methods pursued by the instructor. This present manual is the outcome of such requests.

My object is to aid the student in his efforts to learn the significance of physical signs and their mode of development.

In the construction of this book I have utilized the results of my own personal experience, as well as the common stock of medical teaching. I have made no attempt to establish pathognomonic or distinctive signs of disease, because it seems to me that precision in diagnosis is more surely attained by treating each sign as subordinate to the various combinations of signs which are found in the different maladies.



I wish to emphasize the importance of knowing the physiological anatomy of the heart and lungs, the relative position of the viscera to the parietes, and the physical signs that can be developed in the normal chest, as upon such a foundation rests the only true basis for a correct understanding of the changes caused by disease.

STEPHEN S. BURT, M. D.

37 WEST THIRTY-SECOND STREET, NEW YORK.

# CONTENTS.

---

	PAGE
INTRODUCTION . . . . .	7
<p>Explanation of the term physical signs; Definition of exploration; Necessity for this method of examination; Importance at the same time of subjective signs; Reason for thoroughness; Faults to be avoided; Chest-marks; Relative position of the lungs, liver, stomach, spleen, and kidneys to the surface of the chest described.</p>	
PHYSICAL METHODS OF DIAGNOSIS . . . . .	17
<p>Six methods enumerated; Calormetation or thermometry in place of succussion; Essential aids to diagnosis; Inspection; Attitude, facial expression, and complexion in health and disease; Deviations in the shape of the chest within physiological limits; Changes in the contour of the thorax resulting from disease, and their significance; Normal and abnormal movements of the chest; Inspection of the sputa as a means of diagnosis; Membranous casts of the bronchi; Rusty sputa; Hæmoptysis; Bacillus tuberculosis; Palpation; Vocal fremitus; Increased by consolidation, abolished by an effusion; Mensuration; Circumference and half-circumference of the chest as affected by disease.</p>	
CALORMETATION . . . . .	31
<p>Basis for the importance of measuring the temperature; Normal heat of the body, and the influence of age and sex thereon; Variations of temperature and their significance; Mobility of temperature in children; Absence of febrile</p>	

	PAGE
manifestations in old persons ; The thermometer and its manipulation ; Temperature in disease ; Bronchitis ; Pneumonia ; Pleurisy ; Hæmoptysis ; Acute miliary tuberculosis ; Chronic phthisis pulmonalis ; Cancer of the lung ; Asthma ; Emphysema ; Edema ; Endocarditis ; Pericarditis ; Chronic heart-disease ; Table of temperatures.	
PERCUSSION . . . . .	40
Definition of percussio ; Foreible and gentle strokes ; Immediate and mediate percussio ; Plexor and pleximeter ; Position ; Manner of percussing ; Chief difficulty ; Acoustics ; Quality, pitch, intensity, and duration ; Recapitulation ; Resonance ; Types of resonance ; Key-note ; Regional percussio in health ; Resonance modified by age, sex, quantity of overlying tissue, and by respiration ; Auscultatory percussio ; Respiratory percussio ; Percussio in disease ; Relative significance of a single sign ; Recapitulation.	
AUSCULTATION . . . . .	58
Reason for the value of auscultation ; Course to be pursued ; Immediate and mediate auscultation ; Necessity for the latter method of listening to the heart ; Stethoscopes ; An experiment that demonstrates the dual function of the ears ; Selection and adjustment of the stethoscope, and suggestions in regard to its use ; Auscultatory signs in health ; Auscultatory signs in disease ; Mode of transmission of bronchial breathing ; Recapitulation ; Adventitious signs ; Harsh breathing defined ; Sibilant and sonorous breathing ; Rhythm of sub-crepitant râles ; Gurgling râles ; Source of the crepitant râle ; Succussion ; Metallic tinkle ; Friction ; Extraneous sounds to be eliminated ; Recapitulation ; Vocal resonance ; Greater velocity of low-pitched tones ; Amphoric voice ; Ægophony ; Pectoriloquy ; Recapitulation.	
DIAGNOSIS BY PHYSICAL SIGNS OF DISEASES OF THE LUNGS . . . . .	82
Importance of acquiring a knowledge of the general principles of physical diagnosis ; Acute catarrhal bronchitis ;	



Chronic catarrhal bronchitis; Summary, signs of bronchitis; Bronchiectasis; Acute capillary bronchitis; Summary, signs of capillary bronchitis; Croupous bronchitis; Asthma; Points of difference between laryngitis, œdema of the glottis, and asthma; Summary, signs of asthma; Hæmoptysis; Unjustifiable risk in disturbing the patient; Pulmonary emphysema; Summary, signs of emphysema; Pulmonary œdema; Summary, signs of œdema.

## PNEUMONIA . . . . . 98

Seat and extension of the affection; Termed single, double, and central, according to its locality; Divided into three stages with respect to the physical signs; First stage, congestion; Rusty sputa; Expectoration sometimes absent in children; Temperature in pneumonia; Temporary flights of the index; High fever in children; Critical conditions of old persons in pneumonia with little if any fever; Crepitant râles overlooked; Second stage, solidification; Occasional absence of bronchial breathing due to feebleness of the respiration; Bronchial puff; Signs of pneumonia in very old persons often obscure; Third stage, resolution; Purulent infiltration; Abscess; Lobular pneumonia; Summary, signs of pneumonia.

## PLEURISY . . . . . 106

Fibrinous exudation; Reason for the occasional absence of friction, and an expedient to favor its production; Differentiation of the first stage of pleurisy from the corresponding stage of pneumonia; Serous effusion; Absence of fremitus; Low range of temperature; Curvilinear flatness; Sense of resistance to percussion-stroke; Explanation of the cause of occasional vesiculo-tympanic resonance above the fluid; Acoustic law of sound-waves; Bronchial breathing in pleurisy; Value of listening in the axillary region; Ægophony; Active distinguished from passive effusion, and from consolidation; Purulent effusion; Aspirator; Thoracentesis; Absorption; Points of difference between the signs of thick, plastic exuda-

	PAGE
tion and of fluid effusion; Adhesions; Summary, signs of subacute pleurisy, contrasted with those of pneumonia; Hydrothorax; Pneumothorax; Pneumo-hydrothorax; Pneumo-pyothorax; Change in the level of flatness; Succussion; Summary, signs of air and fluid in pleural sac.	
<b>PHTHISIS PULMONALIS</b> . . . . .	125
Acute phthisis; Chronic phthisis; Divided into two stages; First stage: The value of "myoidema" as a sign; Caution about the early signs of phthisis; Second stage: Various evidences of excavation, and where to look for them; Summary, signs of chronic phthisis; Acute miliary tuberculosis; Fibroid phthisis or interstitial pneumonia; Distinguishing feature, retraction of chest; Sense of resistance on percussion; Pulmonary gangrene; Differentiated from fetid bronchitis, and from phthisis with gangrenous odor; Cancer of the Lung; Points of difference between the flatness of a tumor and that of fluid effusion.	
<b>EXPLORATION OF THE HEART</b> . . . . .	140
Location of the heart; Relative position of its borders to the surface of the chest; Superficial cardiac space; Arteries; Aorta; Pulmonary artery; Arteria innominata; Site of valves; Heart-sounds and where heard; Sounds in contradistinction to murmurs; Mechanism of the former; Circulation of the blood through the heart; Methods of examination; Normal position of the apex; Various causes for its displacement and the significance thereof; Meaning of strong and of weak impulse; Thermometry in cardiac disease; Percussion; Dullness and flatness of this organ; Auscultation; One cardiac revolution; Analysis of heart-sounds; Mapping the superficial space by vocal resonance; Sounds modified by disease; Accentuation of the pulmonary second sound.	
<b>HEART-MURMURS</b> . . . . .	154
Definition; Cause demonstrated by an experiment; Loud and soft murmurs; Anæmic bruits; Murmur accompanying	

a lesion the rule; Result of valvular disease upon the cavities of the heart; Nature's compensation; Danger-signal; The quality of a murmur and its import; Question presented; Genesis of a bruit; Effect of position and of medication upon a murmur; Impact of heart against the lung a source of error; Systolic murmurs; Possible bruits; Determining a murmur; Anæmic bruit; A transient dynamic murmur; A temporary tricuspid bruit; Murmurs that will disappear; Diastolic murmurs; Exceptions; Presystolic murmurs; The same in connection with aortic regurgitation; Pericardial friction; Associated murmurs; Venous bruits; Distinguished from arterial; Hypothesis regarding the venous hum.

#### DIAGNOSIS BY PHYSICAL SIGNS OF DISEASES OF THE HEART AND OF THORACIC ANEURISM . . . . . 170

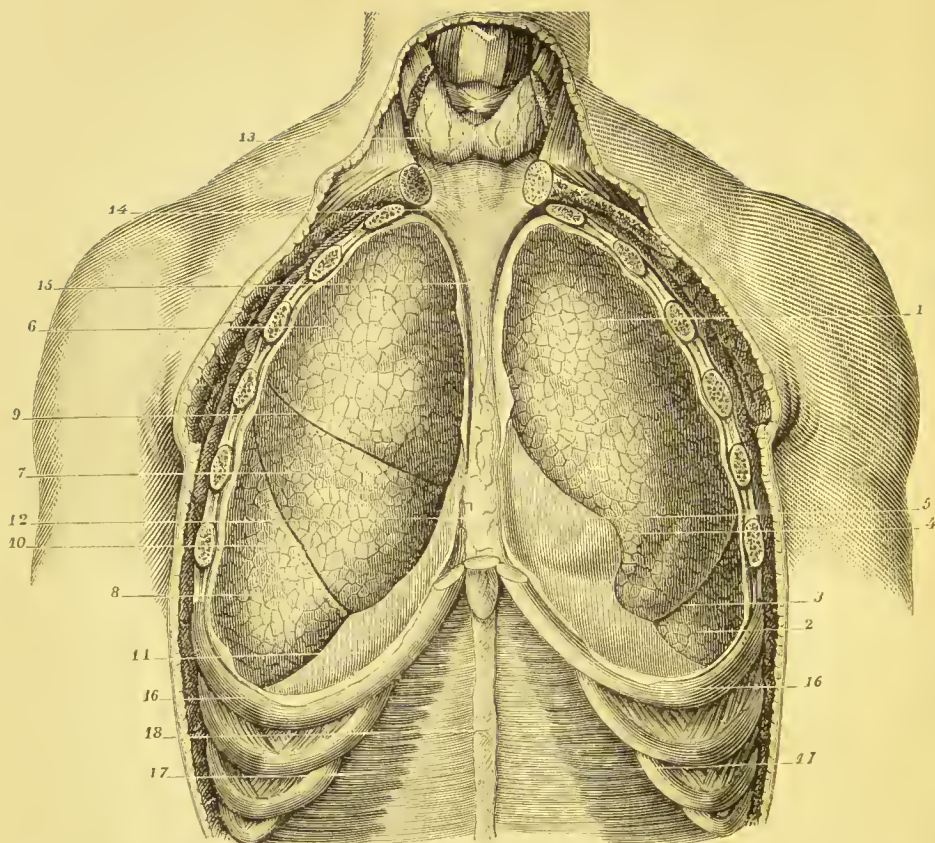
Endocarditis; Complications; Ulcerative variety; Curable murmurs; Static and progressive lesions; Pericarditis; Discovered only by physical exploration; Adhesions; Signs elusive; Hydropericardium; Cardiac hypertrophy; Dilatation of the heart; Fatty degeneration of the heart; Its bearing compared with valvular affections upon surgical anaesthesia; Cheyne-Stokes respiration.

#### AORTIC STENOSIS . . . . . 182

Differentiation of the murmur of this organic lesion from functional bruits; Aortic insufficiency; The significance of Corrigan's pulse therein; Mitral stenosis; Mitral stenosis with insufficiency; Useful hints for separating the two murmurs of this lesion; Commonly one murmur, and the reason; Delay in the radial pulse; Mitral insufficiency; Effect of pulmonary emphysema upon the heart and its murmurs; Tricuspid insufficiency; Venous reflux; Thoracic aneurism; Location; Expansive impulse; Pressure signs and symptoms.







LUNGS, ANTERIOR VIEW (SAPPEY).

1, upper lobe of the left lung ; 2, lower lobe ; 3, fissure ; 4, notch corresponding to the apex of the heart ; 5, pericardium ; 6, upper lobe of the right lung ; 7, middle lobe ; 8, lower lobe ; 9, fissure ; 10, fissure ; 11, diaphragm ; 12, anterior mediastinum ; 13, thyroid gland ; 14, middle cervical aponeurosis ; 15, process of attachment of the mediastinum to the pericardium ; 16, 16, seventh ribs ; 17, 17, transversales muscles ; 18, linea alba.



# EXPLORATION OF THE CHEST.

---

## INTRODUCTION.

THE term physical signs, in medical parlance, refers to certain objective conditions of the body, as distinguished from what are commonly known as subjective signs or symptoms.

Exploration is the careful examination of these physical signs in their manifestations of health as well as disease. From repeated observations of patients, there was gradually evolved a system of signs, which bore a close relation to the condition of the organs as they were found upon post-mortem investigation. And, inasmuch as diseases of the chest have many symptoms in common, there is a manifest necessity for this objective manner of procedure. But, though we shall dwell here more especially upon these methods, nevertheless, it must be kept in mind that the subjective signs hold a position of considerable importance in the solving of medical problems.

The sciences are but the development of common sense in special directions ; and the science of physical diagnosis forms no exception to this rule. If the student will diligently apply himself to the principles involved in this subject, providing his senses are fairly acute, there is no good reason why he should not attain satisfactory proficiency therein. All examinations of the thorax should be exceedingly thorough and methodical, so that no part by any chance shall escape attention. Each student should be honest with himself, and never claim to hear a sound when not certain that he does hear it as described to him. It is a matter of frequent remark, among those best able to judge, that some students are prone to acquiesce too readily in a diagnosis made for them. They are apt to hear whatever they are told to hear, and do not take the requisite trouble to positively assure themselves. This is a fault, and should be avoided, as little real progress is made under such circumstances.

It is well established that mental or nervous vibrations, which are at first the result of close and studied attention, ere long become facile and automatic by repetition. What is known as intuition is but the rapid action of an educated nervous system. A novice, therefore, should not emulate an experienced ex-

aminer in this apparently easy process, for such emulation would soon resolve itself into the merest guess-work. On the contrary, he should endeavor to be slow and painstaking in the extreme. In fact, it is always better to submit to the tedium of methodical examination than ever to risk the danger of an erroneous diagnosis.

For the convenient reference of the student, the following description of the relation which the viscera bear to the surface of the body is given. And the outlines of these organs should be traced upon the chest of some available person in order to fix them in the memory. This is best done with one of those little cosmetic pencils that an actress employs in coloring her eyelids, and which is obtainable at almost any shop where toilet articles are sold.

In order to designate the position or extent of the various thoracic signs, some chest-marks are required, and the sternum, ribs, clavicles, and scapulæ, together with a few artificial vertical lines, will adequately supply this requirement. These lines are as follows—viz.: one falling from the middle of the clavicle to the free border of the ribs, known as the *mamillary* line; one midway between this and the edge of the sternum, called the *parasternal* line; one from the apex of the axilla, the *axillary* line; and one from

the lower angle of the scapula, termed the *scapular* line.

**Lungs.**—The trachea, passing down behind the sternum, divides on a level with the second rib where the manubrium joins the gladiolus. To the right the

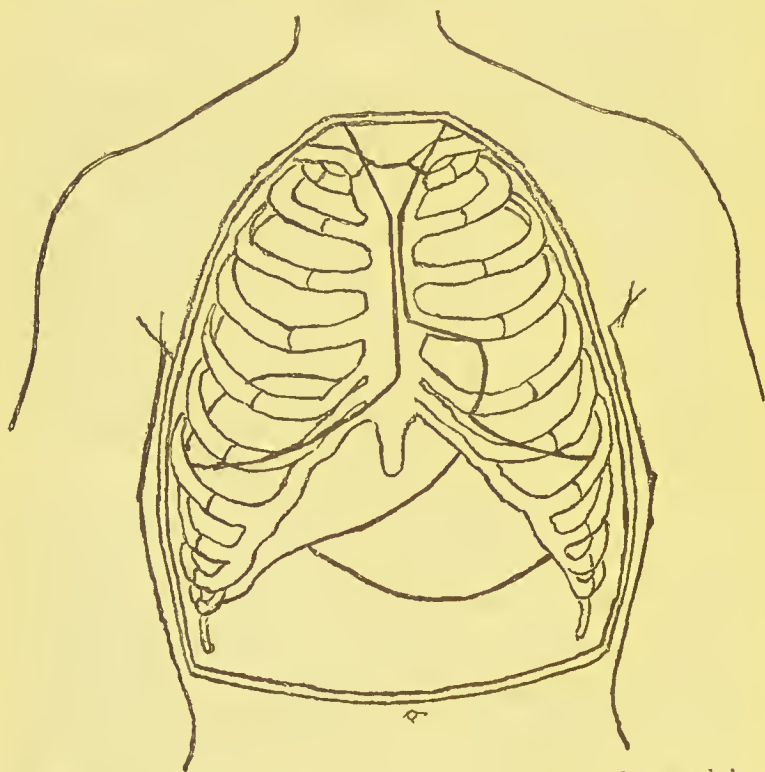


Diagram illustrating the borders of the lung, liver, and stomach in front.

main bronchus lies behind the second costal cartilage, to the left the main bronchus is a little below the car-

tilage. These subdivide into two branches before entering the lungs.

The lung, in front, rises from one to one and three quarters of an inch above the clavicle, usually somewhat higher on the right than the left side. The anterior borders of this organ pass obliquely downward and inward across the sterno-clavicular articulation to the junction of the manubrium with the body of the sternum. From this point they are in contact down to the fourth rib. Here they separate. The right lung extends to the sixth intercostal space in the median line, where the lower border turns to the right along the cartilage of the sixth rib, and downward to the seventh rib in the axillary line, the tenth rib in the scapular line, and the eleventh rib between the scapular line and the vertebral column.

The anterior border of the left lung turns to the left along the fourth costal cartilage as far as the parasternal line, and thence down to the fifth costal cartilage. Here it curves downward and inward to the sixth costal cartilage, and then again to the left, which leaves an open space for a portion of the heart and a tongue-like projection over the apex. The lower border runs along the lower margin of the sixth rib, and down to the seventh rib in the axillary line, the tenth

rib in the scapular line, and the eleventh rib between this and the vertebral column.

The upper borders curve backward from the apices to the spine of the seventh cervical vertebra, while

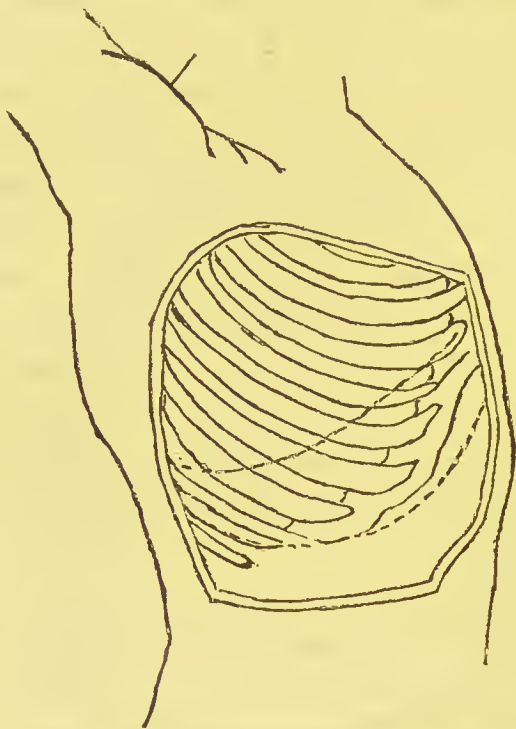


Diagram showing the right lower border of the lung, and liver.

posteriorly the borders run downward to the eleventh rib, one on each side of the vertebral column.

The interlobular fissure on the left side extends from the lower border of the lung at the mammillary



line, upward and backward to the base of the spine of the left scapula. Upon the right side are two fissures, one from the lower border of the lung near the mammillary line, upward and backward to the base of the spine of the right scapula; the other from the anterior border of the lung at the fourth costal cartilage, backward and slightly upward, joining the former a little above the inferior angle of the scapula.

**Liver.**—A greater part of the liver lies upon the right side of the body, just beneath the diaphragm; its left lobe extends about two inches to the left of the median line.

The upper border corresponds to the arch of the diaphragm; the lower border to the eleventh rib behind, the tenth intercostal space in the axillary line, and the tenth rib at the mammillary line; whence it passes obliquely upward and inward from under the right costal arch, and crosses the median line, nearly three inches below the ensiform cartilage, to the left costal arch.

The liver, at its highest point, reaches the fourth intercostal space, and its convex surface approaches sufficiently near the chest-wall to be demonstrable, by percussion, at the fifth rib in the right nipple line, and at about the fifth intercostal space in the right axillary line.

**Stomach.**—The main part of the stomach is situated in the left upper abdominal region. Its pyloric portion extends across the median line beneath the liver to the right costal arch. The upper surface rises to about the fifth rib in the left mammillary line. Its

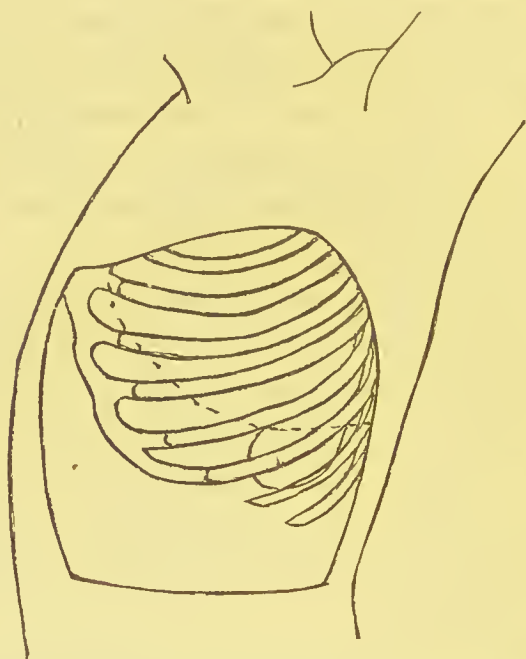


Diagram showing the spleen and the lower border of the left lung at the side.

lower border or great curvature, passing downward and inward, emerges from the costal arch at the level of the tenth costal cartilage, and crosses the median

line midway between the umbilicus and the ensiform cartilage.

Although the stomach retains its location in the left upper portion of the abdomen, yet the relative position of the borders of the organ to the surface of

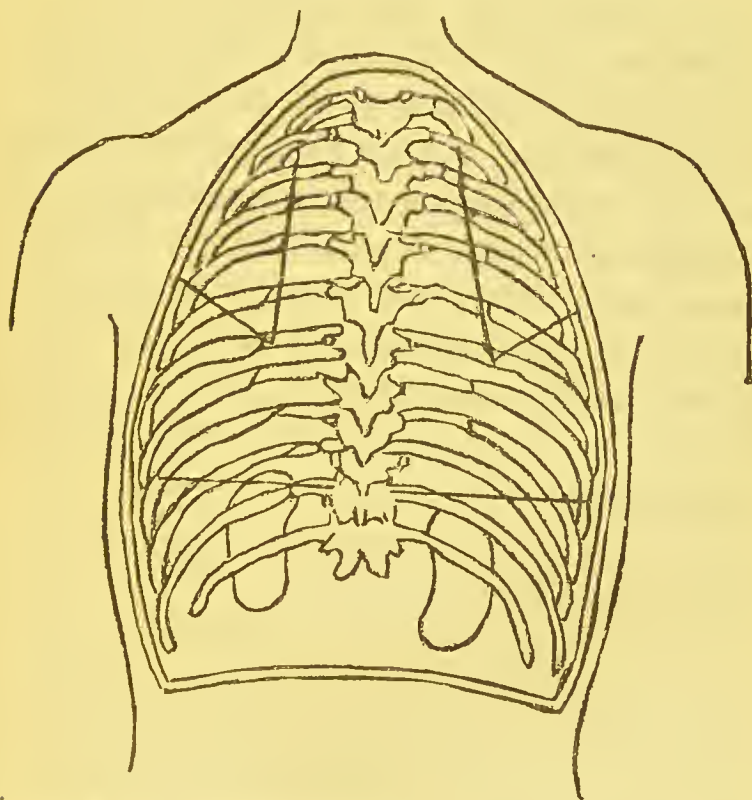


Diagram showing the kidneys, and the lower border of the lung behind.  
the body is modified by the nature and amount of its contents.

**Spleen.**—The spleen is found in the left hypochon-

driac region, just beneath the diaphragm, between the posterior border of the stomach and the left kidney. It extends from a little above the free border of the ribs, at the left axillary line, obliquely upward and backward under the lung. About one third of its surface is covered by pulmonary tissue.

**Kidneys.**—The kidneys are situated in the posterior part of the abdominal cavity, one on each side of the spinal column. The left kidney, which is in contact at its upper extremity with the posterior border of the spleen, reaches from the superior margin of the eleventh rib downward nearly to the crest of the ilium. The right kidney, which at its upper end is beneath the liver, passes from the inferior margin of the eleventh rib downward, and is thus somewhat lower than the left.

## PHYSICAL METHODS OF DIAGNOSIS.

IN accordance with an idea of thoroughness, six methods of examination are laid down, and, though not of equal value, yet each has its special sphere of usefulness. It is too often the custom to disregard all means of physical exploration except those of auscultation and percussion, but the writer hopes to prove that in many ways these somewhat neglected methods are worthy of consideration.

The methods of examination here to be used are, with one exception, the same that are ordinarily given—namely, inspection, palpation, mensuration, calormetation, percussion, and auscultation.

The term calormetation, a compound of two Latin words—*calor*, heat ; *metatio*, measure—is adopted in order to displace succussion, and at the same time conform as nearly as possible to the established nomenclature. For, though succussion usually occupies a position among the methods of exploration, its applicability is extremely limited ; whereas calormetation

or thermometry receives comparatively little attention, while it has a wide range of utility. Succussion, which is practically but a form of listening, will be described together with auscultation.

The more essential of the auxiliaries to diagnosis are the stethoscope, thermometer, plessor and plessimeter, graduated tape, microscope, and aspirator. These will be explained, so far as necessary, in connection with an account of the methods in which they are employed. Among the less needful aids may be mentioned the cardiometer, stethometer, and sphygmograph.

#### INSPECTION.

The natural commencement of an examination for thoracic disease is by inspection, and the information thus obtained is by no means unimportant. It consists in carefully *looking* at a patient, and looking with some definite idea of what appearance a person in health should present, as well as of what are the numerous variations incident to disease. Attention is first directed to the facial expression, the position assumed by the patient, and the manner of his breathing. If pain is depicted on his countenance, the mind of the examiner immediately reverts to its probable cause. He thinks of acute pleurisy, of the pleu-



ritic stitch that may accompany pneumonia, of intercostal neuralgia, of rheumatism, and of angina pectoris. Experience teaches many fine distinctions between these different expressions of suffering. One glance may show the anxious, pale countenance, the rapid, shallow, catching respiration, the inclination forward and to the affected side, of a sufferer from acute pleurisy. The face in pneumonia is pale, with a dusky flush upon the cheek; the breathing is very much increased in frequency; there is rapid, panting respiration, either with or without dyspnœa; and the posture of the patient is upon the side or the back. In angina pectoris there is a deathly pallor, sweat stands upon the face, the look is that of agony, and the victim seems nailed to the spot where the pain seized him. Acute capillary bronchitis produces an appearance of extreme distress; the face, bathed with perspiration, is livid, the lips and finger-nails are cyanotic, and there is marked dyspnœa with frequent orthopnœa; it being impossible for breathing to continue except in the upright position.

During a paroxysm of asthma there is a most imploring, terrified expression upon the pale or flushed face. With open mouth, dilated nostrils, and sweat starting from every pore, the ill-fated object of these seizures fairly gasps for breath, as he clings to the

nearest support that will aid him in his frantic efforts to avoid suffocation. In strong contrast are the pallor, hectic flush, strange luster of the eye, emaciation, and placidity, if not buoyancy, of the tuberculous subject. Clubbed finger-tips and incurvated nails may be found in these cases; and such a condition of the fingers, though existing in other thoracic affections, and not always in phthisis, is yet very frequently seen in this disease. The emphysematous patient presents a dusky, dejected countenance, full face, thick nostrils, and prominence of the auxiliary respiratory muscles upon a rather emaciated neck. His attitude is stooping, his breathing slow, labored, and very much prolonged. Those affected with heart-disease display a bluish-red color in the lips after a little unwonted exertion, which gives early warning of an impaired circulation. And when the balance between the venous and arterial systems is much disturbed, through failure of compensatory hypertrophy of the heart, the dark-red color becomes well-marked cyanosis, and cedema appears around the instep. Cedema, too, is a frequent attendant upon the last stage of phthisis.

Anæmia is shown in a permanent pallor of the face or rather of the inner aspect of the lips, for a bright flush not uncommonly pervades the cheek.

Fullness and congestion of the face, with visible pulsation of the carotid and brachial arteries, may be found in cardiac hypertrophy. Regurgitation at the tricuspid orifice frequently results in pulsation of the right jugular vein. Dilatation of a pupil may tell of a slight and contraction of a strong aneurismal pressure upon the nerves that come from the cilio-spinal center.

Turning, finally, to the chest, it is better if possible to have the covering wholly removed, or from that part under immediate observation, so that the slightest inequalities may be seen. Place the patient upon a plane surface if he is to be examined while lying down, and, if either standing or sitting, arrange his body so that the shoulders shall be on a level. Then look attentively at the form and movements of the chest, which should be viewed from all possible directions. A perfectly symmetrical chest is somewhat uncommon, but not so rare as to require especial description. Reference will be made, however, to a few of the slight deviations that occur within physiological limits, and also to one not infrequent deformity, for all irregularities due to extrinsic causes must be distinguished from those that are the result of both old and recent thoracic disease. The alterations in symmetry are expansion, retraction, bulging, and de-

pression. Bulging is a localized expansion; depression, a limited retraction of the chest. There may be more or less bulging of the lower portion of the thorax on the right side behind and on the left side in front.

Depression is often found at the lower end of the sternum, and sometimes in the left costal arch. These, together with slight deviations of the dorsal spine to the right and of the sternum to the right or the left, may still be physiological.

The commonest distortion of the thorax comes from a condition known as pigeon-breast, in which the sides of the chest are compressed and the sternum thrown forward. As a result of this, the customary relation of the viscera to the surface is destroyed and the accuracy of an examination impaired.

Now, under what circumstances are changes in the shape of the chest found resulting from thoracic disease? They are as follows: A very abundant pleuritic effusion produces expansion of the affected region. Emphysema (vesicular) results in expansion of the whole chest, which is, however, commonly more marked upon one side. In pneumothorax there is a unilateral expansion of the upper portion of the thorax. Bulging may occur over the region of the liver, spleen, or heart from enlargement of these or

gans. It may be also the outcome of aneurismal or other intrathoracic tumors, of pericardial effusion, or of circumscribed pleurisy.

Retraction of the chest upon one side frequently follows absorption of a long-existing pleuritic effusion. Unilateral or possibly bilateral retraction is due to chronic interstitial pneumonia or fibroid phthisis. In narrowness of the antero-posterior diameter of the thorax, with deflection of the sternal end of the clavicle downward, is found an inviting condition for tubercular deposit at the apex of a lung. Depression above and below the clavicle in an otherwise normally shaped chest may be one of the signs of phthisis. Localized depressions of the chest-wall, more apparent with inspiration, are significant of pleuritic adhesions.

**Movements of the Thorax.**—When we observe the respiratory action of a healthy person who breathes quietly, there is seen first an expansion of the lower part of the chest, elevation of the upper portion, and protrusion of the abdomen, which comprise inspiration. Following this comes a return of the chest-wall to its former position, together with a subsidence of the abdomen, which constitute the act of expiration.

It will be noticed that the sexes differ in point of

maximum freedom of respiratory movements. The upper portion of the thorax takes a much more active part in women, whereas the seat of greatest mobility with men is at the lower third and in the abdomen. Superior and inferior costal are the terms applied respectively to upper and lower chest breathing, while the abdominal play is termed diaphragmatic respiration. During labored breathing less difference obtains between man and woman. Respiratory movements are from eighteen to twenty per minute in healthy adults. They are more rapid in women than in men, and in children than adults. Both excitement and exercise increase the respirations. As a result of disease they are either increased, diminished, or labored. Where the ratio is greater than one respiratory act to four cardiac pulsations, pulmonary lesions may be suspected. Breathing is increased by pleurisy, phthisis, pneumonia, and bronchitis of the smaller tubes. It is frequently diminished to avoid pain, as in the first stage of pleurisy, in intercostal neuralgia, and in pleurodynia. Breathing is less frequent on the side of the chest that contains a fluid effusion and more rapid than normal upon the other side. Respiratory action becomes labored in emphysema, asthma, and acute capillary bronchitis; and, while either diminished or absent in hydropneumothorax on



the affected side, it is also labored as well as diminished on the unaffected side.

Thus inspection narrows the possible state of affairs down to a few conditions which, if somewhat closely resembling one another, can be differentiated by the test of other methods. And this exemplifies what holds throughout physical exploration. One sign is never to be trusted for diagnosis, but rather the combined testimony of several signs.

**Sputa.** — Extending the investigation from the chest to the material expectorated may prove invaluable in diagnosing thoracic disease. As an aid to this examination comes the microscope, whose manipulation, however, must be learned from books devoted to that subject. Blood from a bronchial hæmorrhage is bright red and frothy in appearance.

The expectoration of acute bronchitis, at first frothy mucus and scanty in amount, soon becomes muco-purulent and abundant. That of acute bronchitis affecting the smaller tubes, capillary bronchitis, consists in casts of the small tubes mixed with mucus from the large. When placed in water the mucus floats on the surface, and the casts hang by small, stringy connections beneath.

In chronic bronchitis, though the expectoration

may be either profuse and watery, or else scanty, sticky, and perhaps streaked with blood, yet more commonly it is muco-purulent, green or yellow in color, and of varying quantity.

With fibrous bronchitis little masses of fibrin tinged with blood are expelled, which, dropped into water, prove to be membranous casts of the bronchi. Some of these casts are cylindrical and others are branch-like in form, and their presence determines the nature of the bronchitis.

Œdema of the lungs is attended by a profuse, frothy, serous expectoration. Congestion, which is usually associated with more or less œdema, produces blood-stained, frothy mucus.

The material thrown out from cancer of the lungs resembles red-currant jelly.

The expectoration of phthisis varies both in appearance and amount. At first a meager, glairy, frothy mucus, here and there dotted with purulent matter, it ere long becomes muco-purulent and copious, and in the end possibly purulent and excessive. It may, too, be somewhat streaked with blood. Fragments of elastic tissue found upon microscopical examination indicate a destruction of lung-substance. The *Bacillus tuberculosis*, a species of bacteria, is found so constantly in the sputa of phthisis under the

microscope, that, while its absence does not necessarily preclude the existence, its presence is very significant of the disease.

Scarcely less characteristic than the casts of fibrinous bronchitis are the sputa of pneumonia. In fact, they become one of the most reliable signs where the pneumonic process is central; still, it must not be forgotten that inflammation of the lung proceeds now and then without expectoration. During the first two days the sputum is a glairy, frothy mucus, which gradually becomes extremely viscid—so tenacious, indeed, that it is expectorated often with difficulty. If deposited on a piece of paper for inspection, it assumes a nearly globular shape, and is found to be a gelatinous, translucent body, sometimes surrounded by a zone of frothy mucus. Turning the paper upside down will not dislodge this sticky material. It varies from a faint amber to a brick-dust color, according to the amount of blood with which it happens to be tinged. This is known as *rusty sputa*, and is peculiar to pneumonitis. As the disease advances, the sputa become less sticky, the brick-dust color gradually disappears, and, finally, with resolution there is a moderate muco-purulent expectoration. But if the malady terminates in suppuration, the matter expelled will be abundant and purulent. And when the sputa are

dark brown in color, watery, and diffuent, they pre-  
sage an unfavorable termination to the disease.

#### PALPATION.

The method known as palpation consists in the gentle application of the palmar surface of the fingers to the body of the patient ; and when the thorax is under examination, this should be simultaneously performed upon both sides, for the sake of comparison.

In doing this, not only the form, size, and movements of the chest can be appreciated, but also the vibrations of the patient's voice, as they are conducted through the lungs to the hand. It is this last function of palpation which constitutes the chief usefulness of the procedure. These vibrations, termed normal *vocal fremitus*, are most pronounced upon the right side of the chest, and especially from the clavicle down to the third rib. Fremitus is regulated very much by the quality of the voice, as well as by the thickness of the parietes. For, while appreciable in the lower register of an adult, it may be entirely absent from a treble voice ; and, though easily felt through a thin chest, a large deposit of adipose tissue will interfere with its transmission.

Vocal fremitus may be increased, diminished, or

absent, as a result of thoracic disease. Whatever increases the density of the lung, as compression, or the consolidation of phthisis and pneumonia, augments it. Now and then fremitus is not only not increased, but is even absent, with extensive solidification. And this sign is nullified by the interposition of a plastic exudation or of normal pulmonary tissue between the surface and the consolidation.

When there is either air or fluid between the lung and the parietes of the pleural cavity, the fremitus decreases, or wholly disappears. For instance, it is not found in pneumothorax, subacute pleurisy, empyema, hydrothorax, nor hæmothorax. Nevertheless, this rule is not absolute, as fremitus will continue in some instances despite a large effusion.

By the application of the hand in pleurisy one can often detect friction, which the rubbing of the pulmonary against the parietal pleura produces. This is known as friction fremitus. And likewise in bronchitis the vibrations of sibilant and sonorous breathing are sometimes felt. This is called rhonchal fremitus.

The respirations are counted in men by resting the finger-tips upon the abdomen, and in women upon the superior costal region—because the mobility of

the chest is more marked at this place in women, while in men, on the other hand, breathing is chiefly diaphragmatic.

#### MENSURATION.

There are numerous ingenious contrivances for measuring various areas of the chest, but for practical purposes a graduated tape will suffice.

Two measurements are commonly made—one of the circumference, on a level with the sixth costal articulation, the other of the semi-circumference, at this same height. For accuracy, a mark should be drawn with the cosmetic pencil, at the median line, in front over the sternum and behind over the spinous process.

An average adult thorax measures thirty-three inches around, and the right side is usually half an inch the larger. With left-handed persons this side is found to exceed the right in size. Hence an allowance must be made for this physiological inequality when either abnormal enlargement or reduction is suspected.

The expansion of the chest during ordinary inspiration is about one quarter of an inch. Between forced inspiration and extreme expiration, however, it is from two and a half to four inches.



We have already dwelt upon the diseases that diminish expansion. They are such as result in adhesions of the pleura with contraction of the lung, in effusions of the thoracic cavity, in consolidation of the lung, and in extensive dilatation of the air-cells. A large effusion would decrease the inspiratory expansion of the involved side, and at the same time increase its semi-circumference; while hepatization of the lung would also lessen expansibility, but not increase the measurement of the affected side.

Furthermore, adhesions of the pleura with retraction of the chest diminish both movement and semi-circumference.

The circular measurement of the lower part of the chest in vesicular emphysema is sometimes decreased by a full inspiration, and higher, at the same time, there is very little expansive mobility.

#### CALORMETATION.

The importance of measuring the temperature, as a method of physical examination, is based upon the fact that in healthy adults the heat of the body maintains itself at about the same daily average under all circumstances; whereas, during disease, in many instances, the temperature not only varies, but also pur-

sues a somewhat characteristic course. Although the presence of a normal temperature does not insure the absence of disease, a persistent elevation above the normal, or a like depression below the established average, would be considered an abnormal manifestation; and, furthermore, sudden elevations or abrupt depressions of temperature are often the harbingers of momentous crises. Conjoined with other signs, an isolated observation may serve to exclude the existence of certain conditions; but, ordinarily, it requires repeated investigations at stated intervals to reach satisfactory conclusions.

The average temperature of a healthy adult, when properly taken in the axilla, is  $98.6^{\circ}$  Fahr., and the daily fluctuations do not much exceed one degree. In children the average temperature is higher and the diurnal variations are greater, there being often a fall of one, two, or even three degrees during the latter part of the twenty-four hours. Women, and especially those having nervous temperaments, somewhat resemble children in mobility of temperature.

Within the rectum or the vagina the mercury rises nearly a degree higher than at the axilla, while in the mouth the index falls a little short of axillary heat. A permanent elevation above  $99.5^{\circ}$  Fahr., or a continuous depression below  $97.5^{\circ}$  F., in the axilla,

signifies something abnormal; and an increase of two degrees from  $98.6^{\circ}$  F. is a pretty certain indication of fever. Temperature averaging slightly above  $100^{\circ}$  F. is found with many chronic maladies. At  $102^{\circ}$  F. fever may be considered moderate, at  $105^{\circ}$  F. severe, at  $107^{\circ}$  F. dangerous; and if maintained for more than a day at  $107.6^{\circ}$  F. it would probably portend a fatal termination to the disease. It is not so much the height as the persistence of the elevation that bodes danger, for in malarial affections there may be a temporary rise to  $106^{\circ}$  F. or even to  $107^{\circ}$  F. without exciting great fear. Patients with temperatures reaching  $110^{\circ}$  F. and higher have recovered. A depression to  $96.8^{\circ}$  F. may be considered alarming, although the region of greatest danger in collapse is thence downward. Children often develop very high temperatures upon slight provocation, whereas old people, on the other hand, frequently reach very critical conditions without the customary febrile manifestations.

With respect to the methods of procedure in thermometrical observations, one may possibly detect the presence of fever by the hand if applied to unexposed parts of the body, but for anything approaching accuracy a clinical thermometer should be employed. The tyro in medicine is often misled by finding face,

hands, and feet cold when a thermometer would register a high degree of temperature either in the rectum, the axilla, or the mouth. Self-registering clin-



Thermometer.

ical thermometers are at all times to be preferred. Such an instrument is composed of a glass tube of capillary bore from four to six inches in length, on which is a graduated scale ranging from  $90^{\circ}$  to  $110^{\circ}$  F. This tube is closed at the upper end, while at the lower end it expands into a bulb which is filled with mercury. Within the canal there is a small section also of this metal separated from the main body by a little air for an index; moreover, a twist in the glass tube prevents a reunion of this index with the remainder of the mercury.

To set the index, let the student grasp the instrument between his thumb and first finger and strike the thenar eminence of one hand upon that of the other until the upper end of the mercury falls below the place on the stem marked normal; but as thermometers are expensive and of necessity fragile, it is well to perform this manœuvre over a bed, should one be at hand, so that if by chance the instrument

slip it will not break upon falling; and while earried in the waisteoat-poecket there is likewise danger that the thermometer will be dropped and broken. This can be obviated by the simple and easy expedient of elosing part of the aperture with a safety-pin.

A thermometer should be tested from time to time with some standard instrument in warm water, and the disparity, if any, between the elevation of the indiees noted.

Whether to put the thermometer into the mouth, the axilla, or the reetum, must be determined somewhat by eircumstanees. Considerable time can be saved by suffieiently warming the bulb of the instrument, before insertion, to raise the mereury to the plaee marked normal.

If the temperature is taken in the mouth, the bulb of the instrument must be held beneath the tongue, the lips kept elosed, and the patient, for obvious reasons, should breath through the nose. If in the reetum, the thermometer should be oiled, and, after the bowels have been emptied, gently introduced. If in the axilla, whieh for many reasons is the most suitable spot, eare must be taken to wipe the plaee dry, to insert the bulb beneath the pectoral fold, and to see that nothing comes between the instrument and the skin. Having attended to these precautions,

let the student draw the arm of the patient across the chest, and retain the limb in this position, in order to close the axilla and to keep it closed. And, in addition, it is well to cover the shoulder with some article of clothing, while at the same time it is important during the observation to make sure that the thermometer remains in place. The time required for an adequate approximation of the index to its point of maximum elevation is from five to seven minutes, providing the above details have been complied with.

The frequency of the observation should be decided somewhat by the urgency of the case, though temperatures are ordinarily taken at least twice in the day, at about eight in the morning and at five o'clock in the evening.

Finally, a record should be made of these fluctuations of temperature, in the form of a chart, which should be preserved for daily inspection.

#### TEMPERATURE IN DISEASE.

**Bronchitis.** — A catarrhal inflammation of the trachea and bronchi may produce no alteration in the temperature beyond the regular daily physiological fluctuation. Still, there is often slight fever at the commencement of the disease, and from time to time



during its progress; and children frequently have severe pyrexia with these affections. The intensity of the fever, more especially with an adult, indicates the amount of the inflammation. But the importance of thermometry in bronchitis, however, rests mainly upon the contingent complications, which announce themselves by sudden elevations of temperature.

**Pneumonia.**—In acute lobar pneumonia the temperature usually rises quickly to  $103.5^{\circ}$ – $104^{\circ}$ , or even to  $106^{\circ}$  F. Here it continues, with daily fluctuations of one or two degrees, until defervescence, which is either abrupt or gradual. A rise of the index above  $104^{\circ}$  F. would indicate considerable fever. Still, patients with a moderately high fever often do best in the end. Pyrexia is supposed to continue with the progress of the inflammation, and a sudden increase in the intensity of the fever signifies that a new lobe is invaded, or that some complication has supervened.

The temperature of lobular pneumonia takes an irregular course; the rise is more gradual, the average less high, and defervescence is more protracted, than in lobar pneumonia.

**Pleurisy.**—The range of temperature with inflammation of the pleura is variable. In some instances there will be considerable fever, in others but a mod-

erate amount, while in many cases there will be no febrile manifestation. When there is pyrexia, it usually does not exceed  $103^{\circ}$  F.; and, moreover, it is remittent in type, and has a gradual abatement.

**Hæmoptysis.**—Most pulmonary hæmorrhages that are copious depress the temperature, and when the bleeding is excessive the depression may reach  $96.8^{\circ}$  F., or even fall below this mark. A reaction from the collapse would be followed by a renewal of any pre-existing fever.

**Acute Miliary Tuberculosis.**—A sudden rise of the temperature, not otherwise accounted for, in a case of chronic phthisis pulmonalis, suggests the not unlikely complication of acute miliary tuberculosis. There is, however, nothing characteristic about the range of the fever in this affection, except, perhaps, a rather pronounced matutinal elevation.

**Chronic Phthisis Pulmonalis.** — Pyrexia prevails during the progressive stages of phthisis, so that a daily afternoon appearance of fever, however slight, would increase the weight of testimony in favor of the presence of this malady should diagnosis be uncertain.

Frequently there is no febrile movement for a time, or there may be a rise only in the evening, or

else a morning elevation with a moderate increase toward the night. Death, in some instances, is preceded by a fall, in others by a decided rise of temperature.

**Cancer of the Lung.**—The temperature in pulmonary cancer either remains normal or slightly falls, except during intercurrent attacks of pleurisy, or of pneumonia, when there will be more or less fever.

Uncomplicated asthma, emphysema, œdema, and hydrothorax exist without fever.

**Endocarditis and Pericarditis.**—With an attack of articular rheumatism there may be either endocarditis or pericarditis without an increase of the temperature over what obtains, save that during convalescence the fever remains somewhat higher than in uncomplicated cases; and, too, some time after the joint trouble has disappeared, there may be a decided elevation of temperature caused by a fresh development of valvulitis.

**Chronic Heart-Disease.**—There is no rise of temperature in chronic heart affections. It is upon the supervention of acute attacks or of some complicating disease that fever is generated.

## TEMPERATURE TABLE.

*Normal.*

98·6° F.

37° C.

*Fever.*

Slight.....	100·4° F.	38° C.
Moderate.....	102·2°	39°
Severe.....	105·8°	41°
Dangerous.....	107·6°	42°

*Collapse.*

Slight.....	96·8° F.	36° C.
Moderate.....	95·0°	35°
Severe.....	93·2°	34°
Fatal .....	91·4°	33°

98·6° F. equals 37° C.

100·4°	"	38°
102·2°	"	39°
104·0°	"	40°
105·8°	"	41°
1·8°	"	1°

Multiply centigrade by 1·8° F. and add 32° to obtain Fahrenheit.

## PERCUSSION.

Percussion is the act of striking the body in a certain prescribed manner to determine the relative amount of air and solids therein contained, to detect the presence of fluids, to define the viscera, and to

locate tumors. This method embraces a wide field of investigation, and its proper performance requires both care and considerable practice. Therefore, close attention should be given not only to the subject in general, but also to the details of manipulation.

In the first place, oftentimes a student is found to be rather too literal in his interpretation of "striking the chest," and thereby the patient nearly gets a pummeling. Although forcible blows become necessary upon very thick tissues and over deep-seated solidification, yet, as a rule, gentle strokes, except under these circumstances, are productive of the most satisfactory results.

There are two methods of percussing: one is directly upon the body, which is termed immediate; the other is upon some interposed substance, and is called mediate percussion. The former is mainly confined to exploration of the clavicles, where the bones themselves answer for pleximeters.

A pleximeter or plessimeter is a flattened oval disk, either of vulcanite or ivory, turned up at each end to be conveniently handled.

A plexor or plessor is a small rubber hammer in the shape of a double cone, fastened at the center to a vulcanite handle.

But, notwithstanding these auxiliaries, percussion is commonly practiced with the unaided fingers, and,



Pleximeter.

while requiring greater dexterity, it is in many ways preferable: First, by using the fingers, which are also better adapted to the inequalities of

the chest, much extraneous noise is eliminated; second, the sense of resistance communicated to the



Plexor.

hands is of very great value; and, third, their availability is enhanced by being ever present when needed.

During exploration, when a matter of choice, the person examined should be seated. For percussion in front the body should be erect but unstrained, the shoulders on a level, and the arms hung loosely by the side. The detection of slight dullness is furthered by placing the patient's back against a thin door for the re-enforcement of sound.



For percussion behind, have him cross his arms, put a hand on each shoulder, then elevate his elbows, and slightly incline his head forward.

For percussion at the side, let him clasp his hands over his head.

But if the patient is unable to sit up, see that his body rests upon a plane surface, with his muscles relaxed and limbs placed symmetrically. It is always better to percuss upon bare skin, yet if there be no alternative, which is often the case with women, some unstarched garment can be worn.

The examiner, assuming a position directly opposite the person to be examined, makes a pleximeter either of the index or middle finger by placing its palmar surface firmly upon the chest along a rib or an interspace. He then flexes the index, middle, and ring fingers of the other hand until their conjoined ends present an even surface for the plexor or hammer. With this plexor each blow should be delivered upon the pleximeter vertically from the wrist, with care that the strokes be equable, fairly rapid, and quickly receding. Moreover, he must avoid striking with the finger-nails on the one hand or upon them on the other hand; and the pressure of the pleximeter as well as the stroke of the plexor must

be uniform for like parts on the two sides of the chest.

Light percussion can be performed by the middle finger with the impulse partly from the metacarpophalangeal joint.

Lastly, never percuss at random, nor indifferently as to the breathing, but at all times with an idea of comparing corresponding areas, and at the same relative respiratory act. For there is a normal difference between the resonance developed over a rib and that over an interspace, and likewise between the resonance at the end of inspiration and at the close of expiration. The pitch is higher over a rib than an intercostal space, and upon a held inspiration than a forced expiration.

The difficulty of percussion seems to be in making a clear light stroke from the wrist; many beginners are apparently seized with a *quasi*-ankylosis of this joint. Still, all obstacles can be overcome by patience and application.

Physical diagnosis makes but a slight draught upon acoustics, and the student should have little trouble in grasping these few principles.

Sounds developed by percussion have the distinctive attributes of quality, pitch, intensity, and duration. Now, the ear does not require much cultivation.

tion to distinguish between the musical sounds of a flute and those of a piano, even when the note and pitch are the same. This is because of the different inherent quality in the tones, due in part to the form of the vibrations. So, in percussion, one recognizes pulmonary resonance, flatness, etc. (although not musical sounds), by their innate quality.

It is not difficult to decide between the bass and high treble notes of a piano; the former are low-, the latter high-pitched tones. A similar difference exists in the pitch of sounds evolved by percussing the chest. Pitch depends solely upon the number of vibrations that take place in a given time. Tension, by increasing the rapidity of the vibrations, raises the pitch; relaxation, on the contrary, by decreasing their rapidity, lowers the pitch.

To continue with the piano-forte for illustration, pressure of the foot upon the hard pedal augments the loudness of the note struck, and upon the soft pedal diminishes the sound. This is accomplished by regulating the size of the vibrations; the large produce loud, the small develop soft tones. Here is shown the greater and the lesser intensity of sound.

The intensity or loudness of a percussion note varies with the texture of the thoracic parietes, the

quantity of contained air, and the forcibleness of the blow.

Duration is simply the continuance of sound, and this is exemplified by holding down a piano-key either a longer or a shorter time.

#### RECAPITULATION.

Quality: Distinguishing inherent character.

Pitch: Elevation of tone; depression of tone.

Intensity: Amplitude of vibrations; loudness.

Duration; Time of continuance of sound.

**Resonance.**—The different kinds of sounds developed by percussion are normal, pulmonary, or vesicular resonance; dullness; flatness; tympanitic resonance; vesiculo-tympanitic; and two other modifications of tympanitic resonance, termed respectively amphoric resonance and cracked-pot sound.

It is almost needless to say that a more adequate conception of these sounds can be obtained by listening to them than from any description of them, the fact is so evident. Therefore let the student select a thin, healthy adult, whose thorax is nearly symmetrical, and uncover his chest in a room that has been suitably warmed. Now observe all directions as to manipulation, and percuss in the region from the left

clavicle down to the third rib. Here is evolved normal pulmonary or vesicular resonance, a rather intense, prolonged, low-pitched sound, which has the quality known as vesicular. Moreover, the pitch, intensity, and duration are peculiar to this individual, and serve for a key-note, as it were, to the resonance in other parts of his chest; for it will be found that vesicular resonance varies in pitch and intensity with different persons. Then percuss on the right side in front, just below the fifth rib, where the liver is covered by a thin layer of lung. The aerated lung and the solid liver-tissue combined give a modified vesicular resonance called dullness, which is short, rather high-pitched, and only moderately intense—a quality of resonance that ranges from slight to complete dullness, according to the relative proportions of air and solid material.

Again, percuss in the same region below the extension of the lung, and a short, high-pitched note of slight intensity will be developed. This has the quality known as flatness, for here the sound comes not from the lung containing more or less air, but rather from an almost solid body.

Percussion below the left nipple, in the region of the stomach, if this organ is distended by gas, will produce an intense, prolonged, usually high-pitched

sound, termed tympanitic resonance. Over the intestines, on the other hand, unless greatly filled with air, the pitch of the note will be low, but, whether the pitch be high or low, the quality of this sound remains tympanitic.

Vesiculo-tympanitic resonance, as the name implies, is a combination of vesicular with tympanitic resonance, and this union determines its quality. Hence, with considerable intensity, there are variations of pitch and duration, as either tympanitic or vesicular resonance predominates. Percussion over the apex of the lung, toward the trachea, sometimes develops a mixture of the vesicular resonance, with a tympanitic sound from the trachea—vesiculo-tympanitic. This, however, is much better illustrated by the disease emphysema, in which it frequently occurs.

Cracked-pot sound can be produced by percussing a little below the clavicles in some adults and in many children. It is a chink-like sound, represented by striking the back of one hand, while clasped in the other, upon the knee.

Amphoric resonance is auto-demonstrable by a slap upon the cheek while the lips are closed and the mouth is moderately inflated.

**Regional Percussion in Health.**—Every student that



takes the trouble to follow the directions which are given for learning the different kinds of percussion resonance, will find that he is rewarded for his trouble by a much clearer idea of what these sounds are than could possibly be secured without this objective method. The next step is to make a systematic examination of the normal chest, and thereby map out the regions where vesicular resonance, dullness, flatness, etc., properly belong. First, however, let us consider some of the individual peculiarities, for chests differ materially.

Resonance is modified by age, by sex, by respiration, and by the thickness of the overlying tissue. Therefore, one person can not be taken as a standard for another; the "key-note" must be sounded in each instance.

In youth the thoracic wall is elastic; consequently resonance is lower in pitch and of greater intensity than in old age.

The intensity is usually greater and the pitch lower in women than in men.

A very full held inspiration may put the muscles into such tension, and so inflate the lung, as to raise the pitch, whereas the pitch is lowered at the end of expiration. Still, this disparity is not observable in every person.

That there should be an increase of intensity with a rise in pitch, contrary to what happens in dullness or in flatness, is possibly explained by an elevation of pitch being due, in the one instance, to aëriform tension with resulting hollowness, and in the other to tension from increased density which amounts to solidity.

Where chests are covered by heavy muscles the percussion note is raised, the duration shortened, and the intensity diminished. This is liable to lead an unwary observer to conclude that there is consolidation of lung beneath, when a cautious examiner, by availing himself of comparison, would avoid such a mistake. And in this we find an example of the inadequacy of sensation, unassisted by comparison and experience.

Now, with these few points in mind, let the student gently percuss the lung on each side above the clavicles; and at the same time see that the mouth of the person examined is open, and that the stroke is not directed toward the trachea. Here there is vesicular resonance, but with pitch sufficiently high to constitute moderate dullness.

Upon the clavicles—and immediate percussion is best over these bones—there is vesicular modified by osseous resonance.

Upon the right side in front, from the lower mar-

gin of the clavicle to the fifth rib, normal vesicular resonance is found. The pitch is somewhat higher than on the left side. Thence to the lower border of the lung, where it shelves over the liver, there is dullness, and below, nearly to the free border of the ribs, flatness. But a tympanitic quality is frequently transmitted from the intestines through the lower margin of the liver.

It is a matter of interest, having found the upper line of liver flatness, to note the amount of respiratory play in the lung. This is shown by percussing from that level downward, after a full inspiration while the person examined holds his breath. With a deep inspiration the lung glides in between the pleximeter and liver, and flatness disappears to the extent of the lung's excursion. On the other hand, forced expiration elevates the upper limit of flatness, and thus the extremes of pulmonary mobility are illustrated. The position of the lower border of the lung fluctuates not only with respiration, but also with the posture of the body. It travels an inch or more in calm breathing, so that the exact lower boundary of the organ depends somewhat on the attitude of the person, and very much on the rhythm of the respiratory act.

On the left side in front, from the lower edge of

the clavicle to the third rib, there is vesicular resonance. From the third rib downward, where the lung overlaps the heart, there is dullness, and, as a rule, flatness over the uncovered heart. This region of flatness is affected by the locomotion of the lung, for flatness can be made almost to disappear on full inspiration. Downward from the sixth rib, if the stomach is well distended by gas, there will be tympanitic resonance. -

From the upper margin of the sternum to nearly the fourth costo-sternal articulation there is resonance of considerable intensity, which is in quality peculiar to the sternum; and, though it may extend down the entire bone, yet sometimes, and especially if the person examined be inclined forward, forcible percussion develops dullness.

Over both scapulæ the pitch is high and it amounts to dullness, but not complete dullness, for an underlying consolidation can still be detected by comparing the resonance of the two sides.

Between the scapulæ and the vertebral column there is vesicular resonance.

From the lower angle of the scapula, on both sides, there is vesicular resonance down to about the tenth rib, with flatness thence downward. Here the limit of flatness is influenced by the shifting lower border

of the lung, and the line of demarkation is a little higher on the right side because of the liver.

In the right axillary line there is vesicular resonance down to about the fifth intercostal space, with dullness from here to the seventh rib, and flatness down to the free border of the ribs.

In the left axillary line there is vesicular resonance to the sixth intercostal space; and thence downward, if the stomach be inflated, there is tympanitic resonance. Still, in this area, by gentle percussion, flatness may be developed from the ninth to the eleventh rib, over the spleen; but, if the stomach be full of food, there will be either dullness or flatness where there was tympanitic resonance.

**Auscultatory Percussion.**—Some zealous examiners have undertaken to define the viscera by sounds peculiar to each organ. But, although it is possible for disparate surroundings to somewhat influence resonance, yet the flatness of one viscus will be found not to differ essentially from that of another.

On the other hand, Clark and Cammann greatly added to the facility of defining the viscera by the introduction of auscultatory percussion. This consists in what may be termed supplementary percussion—i. e., a stethoscope is employed to conduct resonance to the ear, while the stroke is delivered by

either the listener or another. An instrument has been made with its objective extremity in the form of a truncated wedge, in order to fit the intercostal spaces, and thus establish closer communication with the underlying substance.

**Respiratory Percussion.**—A system of percussing with the breath of the one examined in suspense, now at the end of full inspiration, and then at the close of forced expiration, is entitled, by Da Costa, respiratory percussion. The efficacy of this plan depends upon the fact that resonance, in many instances, is materially altered by respiration. For example, a full inspiration, when held, not only increases the intensity, but also raises the pitch, while forcible expulsion of the air both lessens intensity and lowers pitch. Neither expedient, however, changes the relative difference between the two sides.

It is the first measure that proves most serviceable, especially when associated with the improvised sounding-board, by bringing into high relief, as it were, the at best slight dullness of incipient phthisis. Moreover, advantage is taken of respiratory percussion to distinguish consolidation from collapse of the lung. For a full and held inspiration often diminishes dullness of the latter, whereas in the former the sign remains unaltered. This applies particularly to bron-



chitis of the smaller tubes, in which dullness may occur from either collapse of the lobules or consolidation of a complicating lobular pneumonia. Furthermore, recourse may be had to respiratory percussion, as a means of definitely determining upon the line of demarkation between a compressed lung and the fluid of a pleural effusion; for an increase of volume in the air of a compressed lung augments the intensity of the resonance, and thereby renders the change to flatness more apparent by contrast.

**Percussion in Disease.**—It is essential, withal, to have some general conception of the physical signs of disease, as demonstrated by percussion, before undertaking the examination of individual cases. Sounds that are normal in one place are abnormal in another place, and evince disease; and, moreover, changes in some of the distinctive attributes of sound have a like meaning, without a change of locality.

Take, for example, flatness, which we found within certain defined areas, over the liver, the heart, and the spleen. Now, an extension beyond these limits may be due either to enlargement or displacement of the viscus, or else to a recession of what overlaps its borders; a falling short, on the other hand, may be from a diminution in the size of the organ, or from some encroachment upon its surface. Again, should

flatness be discovered elsewhere in the chest, it would be one of the signs of a fluid effusion, of entire consolidation, of thick pleuritic adhesions, or of a tumor.

In a similar manner, when tympanitic resonance, normal over the gastric region in health, is evolved from other parts of the chest, it becomes one of the indications of pneumothorax, and possibly of a large air-containing cavity in phthisis. Now and then, too, this resonance can be heard over solidified lung, in which it is generated by the air of the bronchi.

An alteration in some of the attributes of resonance is exemplified when slight dullness, where it exists normally, reaches complete dullness, with a corresponding rise in pitch and decrease of intensity.

The solidification of phthisis and of pneumonia, as well as of compressed lung, gives dullness. There will be dullness from an extensive pulmonary œdema, from a large hæmorrhage in the lung, and from moderate pleuritic adhesions.

Within the area of normal vesicular resonance the pitch may be raised and the intensity exaggerated, from vicarious breathing. This change of pitch and intensity, which practically amounts to vesiculo-tympanitic resonance, is sometimes developed over a lung that is condensed but not collapsed by an effusion. And, moreover, where one lobe is hepatized, such

resonance may be found in the unaffected portion of the lung. It occurs also in extensive emphysema.

Amphoric resonance is heard in pneumothorax and over a large, empty pulmonary excavation with tense walls.

Cracked-pot sound is developed over cavities with flaccid walls. It may escape observation, however, unless the examiner during percussion place his ear in close proximity to the patient's open mouth. This sound is not peculiar to vomicae, for it is audible in some instances over a compressed or a solidified lung and in the chests of many healthy children.

From the foregoing account of the physical manifestations of disease it is seen that a single sign has not so much an absolute as a relative significance; and by keeping this fact in sight physical exploration is made less disappointing than by always looking for signs that are pathognomonic. Every intelligent diagnostician draws his conclusions from a combined testimony not only of signs but of symptoms as well.

#### RECAPITULATION OF RESONANCE.

Vesicular: Uncomplicated lung.

Dullness: Lung with increased proportion of solids.

Flatness: Solids, fluids.

Tympanitic: Large body of air.

Vesiculo-tympanitic : Lung with increased proportion of air.

Amphoric : Empty cavity with tense walls.

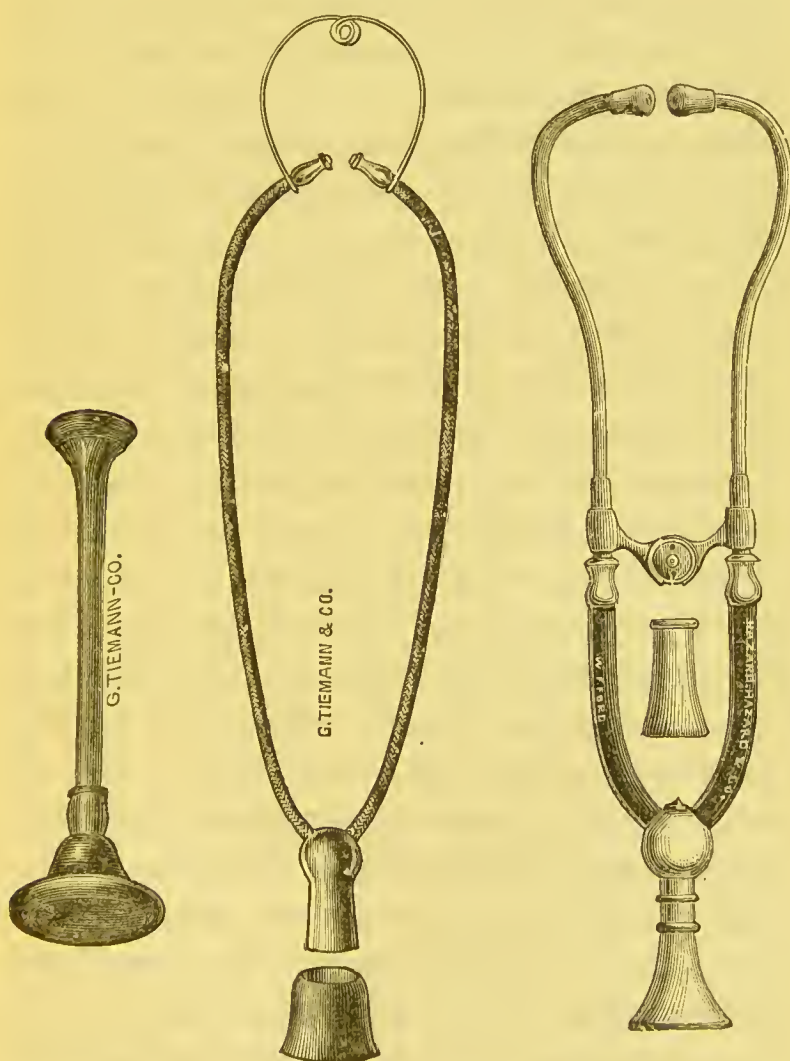
Cracked-pot : Cavity with flaccid walls.

#### AUSCULTATION.

The act of listening is termed in medicine auscultation, and the diagnostic value of this procedure rests upon knowing what should be heard in health as well as what are the variations incident to disease ; wherefore it may be inferred that the study of auscultation of the thorax includes a consideration not only of the abnormal but also of the normal sounds. Moreover, it follows that the course to be pursued with this method should not differ from the one taken with percussion.

There are two ways of listening to the chest, namely, to place the ear upon it, which constitutes immediate auscultation ; and to listen through some conducting substance, which is called mediate auscultation. Of these methods the first is preferable in the observation of respiration, and the second in the detection of heart sounds and murmurs. In fact, with the heart, mediate auscultation is at times indispensable.

Instruments to convey sound from the body are termed stethoscopes. Now, a sound is a vibration



Single inflexible  
Stethoscope.

Flexible double Steth-  
oscope.

Double Stethoscope.

that is appreciable by the ear, and a stethoscope is a medium for the direct transmission of these vibrations from the chest of the person examined to the ear of the examiner. Some stethoscopes are flexible while others are inflexible, and of these two kinds there are many varieties. An inflexible stethoscope usually consists of a slender cylindrical stem six or eight inches in length, with a small disk at one end, the aural extremity, and a slight expansion at the other end, the thoracic extremity; and since elasticity is essential to the molecular vibration of this medium, and the greater the elasticity the greater the facility of transmission, the choice of material should be guided by this qualification. Among hard substances either wood or iron best fulfills such a requirement, and the wood must be cut in the direction of its fiber. We have diverse flexible stethoscopes, including both single and double instruments. One form of binaural stethoscope is composed of a rubber tube for each ear, kept in place by a curved wire spring and inserted in a hollow, somewhat funnel-shaped objective extremity. Another form has a similar thoracic end-piece seven eighths of an inch in diameter by three in length, and a couple of short flexible tubes which are connected by a mortise-joint with two metal tubes curved to fit each ear and held in position by a spring concealed in



the hinge that joins them. In this instrument the wave-sounds that enter the tube are prevented from expanding, hence they reach both ears with undiminished energy. A flexible binaural stethoscope has much to recommend it. The pliability enables the examiner to assume an easy attitude during auscultation, which is very desirable, and at the same time to see that the thoracic extremity of the instrument is properly placed, which is absolutely essential. Perfect adjustment of the auricular ends to the external auditory canals at least shuts out all extraneous noises, if it does not actually increase the perception of sound. Some authorities maintain that there is no good reason why two ears should hear better than one; yet, in spite of that, a double stethoscope seems to magnify the sound. It is believed by others that there is an additional function in the combined use of two ears, locating the direction whence sound-waves proceed. Roosa says that most of those who have lost the use of one ear complain that it is very difficult to decide where sound comes from; and the writer has discovered, in testing the relative merits of a number of stethoscopes, what appears to be a demonstration of this dual function of the ears. The steps of the experiment are as follows: Let the observer take a binaural stethoscope, with arms made of soft rubber

tubing, and listen to the beating of a watch. The sound now will be referred by him to where the watch is. If, however, he close one of the tubes by squeezing it, the tick of the watch will then be not only shut out from this ear, but also transferred from the locality of the watch to the other ear, and the perception of direction lost. Next, a change of pressure to the other tube carries the sound back, not to the watch, but to the former ear. Finally, by a removal of the obstruction from the tube, the beating is heard again in the direction of the watch on which rests the objective end of the stethoscope. This holds, likewise, when one listens to the heart, and the application of so interesting a fact to auscultation is the part it possibly takes in rendering a double more accurate than a single stethoscope. Whether it more sharply defines the position of the sound or in reality increases the intensity, the binaural instrument, by engaging both ears, improves hearing and seemingly augments the sound. That it does magnify sound, moreover, is commonly urged as its greatest objection. But a stethoscope is seldom required in listening to respiration, and, when thus employed, due allowance can be made for such alteration. Furthermore, loud heart-murmurs are easily heard, yet these do not always indicate grave lesions; while exceed-

ingly soft murmurs are difficult to hear, but sometimes accompany serious disease. Hence the very fact that sound is exaggerated by these stethoscopes facilitates the detection of murmurs that might be otherwise overlooked, and renders diagnosis possible where it was previously impracticable.

In the choice of a binaural stethoscope the student should be careful to select one that fits his ears closely, thereby excluding external sounds, but still not so tightly as to cause pain, for this would divert attention from the sensation of hearing. The small objective end should be not more than seven eighths of an inch in diameter, and the spring or elastic joining the arms not too stiff. Always adjust the instrument with the concavity of the metallic tubes in front, so that the ear-pieces shall enter in the direction of the auditory canals, which is inward and forward. Place the thoracic end evenly and firmly upon the chest, and hold the stethoscope between the thumb and one finger.

For auscultation the positions of the person examined are those taken during percussion; the examiner should so place himself as to be free from all constraint; and, though mediate auscultation must be made upon the bare skin, yet the exposure need not be extensive.

The same precautions should be observed in listening as when percussing. Compare similar regions repeatedly, and never fail to include the whole chest in the examination, for a want of thoroughness is quite as disastrous as a lack of skill. It is not uncommon to mistake friction of the stethoscope upon a hairy chest for signs of thoracic disease; also the throat-sounds or noises made by many persons while breathing are liable to a misinterpretation. By directing a patient to open his mouth during the examination, noisy respiration often can be obviated, and, moreover, it is an ingenious device for the prevention of annoying garrulity. Shaving the hair will remove the cause of the first-mentioned complication. Furthermore, the respiratory signs fail in their development where the breathing is shallow; and when this is the case, a few enforced coughs will be followed of necessity by a deep breath and the respiratory sounds. Lastly, some effort is requisite to concentrate the attention upon auscultation, particularly with beginners, and the consequent fatigue soon dulls the sense. A nerve-center becomes exhausted just as overworked muscles do, and when this stage is reached, as it often is through constant listening, the auscultator should temporarily refrain from further work.

**Auscultatory Signs in Health.**—Respiratory sounds

are those of inspiration and of expiration. They are characterized by quality, pitch, intensity, duration, and rhythm. This last refers to the regular succession of the two acts which comprise respiration; the others have been duly explained in connection with percussion.

Now, certain types of breathing can be heard in healthy persons just as many kinds of percussion resonance have been observed. These are vesicular, bronchial, and broncho-vesicular respiration.

An alteration in the character of the breathing and likewise a change of position include some of the important signs of disease, so that one must become familiar with the typical respiration of different localities.

Take, therefore, a person under the same conditions indicated for percussion and perform immediate auscultation in the region on the left, between the clavicle and the third rib. Here is heard a prolonged, low-pitched, soft inspiration, followed without interval by a short, low-pitched, soft expiratory sound. This is vesicular or pulmonary breathing. In the corresponding region on the right, vesicular respiration is somewhat higher in pitch and expiration more prolonged than upon the left; and as the examiner approaches the trachea and larger bronchi either in front

or behind, breathing becomes less vesicular and approximates bronchial respiration.

Inspiration in some instances may be loud and in others quite feeble, while expiration is not infrequently absent. The intensity of the breathing is great during childhood, variable in middle life, and diminishes with old age. Children have what is called puerile, old persons senile respiration. Vesicular breathing, then, subject to these modifications, is heard in the chest wherever there is normal lung-tissue.

If the listener will now place his stethoscope over the trachea, a long inspiratory sound can be heard, high-pitched and intense, like a current of air passing through a tube. This is followed after a short interval by expiration which has the same tubular quality, but a higher pitch, greater intensity, and longer duration. This is known as bronchial or tubular breathing.

As its name implies, broncho-vesicular respiration partakes of the characteristics of two kinds of breathing, and proportionately as one or the other predominates. Here inspiration is usually shortened in duration, raised in pitch, and of increased intensity, while, if not absent, expiration after a pause is prolonged and higher pitched, as well as of greater intensity than



in the first act. In the normal chest broncho-vesicular breathing can be heard where a thin layer of lung overlaps the bronchi, as between the scapulæ behind and also near the large tubes in front.

Cavernous and amphoric breathing are two forms of respiration not found in health. The first is a hollow, low-pitched sound, with a longer and lower-pitched expiration than inspiration; the second is but a modification of the first, and has a musical quality more or less resembling the sound generated by blowing into the mouth of an empty flask.

**Auscultatory Signs in Disease.**—The normal vesicular respiration undergoes sundry alterations in disease. It may be either exaggerated or diminished, or else suppressed. Under some circumstances the rhythm becomes divided, under others interrupted, while expiration is often prolonged; and in the place of vesicular breathing broncho-vesicular or bronchial breathing may be found.

An exaggerated breathing is heard over that part doing extra work for a disabled lung wherein, for instance, there is an extensive consolidation or where the lung is compressed by a large effusion.

A diminished breathing occurs where a moderate fluid effusion or a plastic exudation exists, and also in some conditions of emphysema and of phthisis.

The suppression or absence of vesicular breathing indicates an extensive effusion of fluid or of air in the pleural cavity, and complete obstruction of a bronchus, as well as certain states of phthisis and of emphysema.

What we term interrupted respiration is a breaking of the rhythm into little puffs, and is chiefly inspiratory. This may be caused by pleurodynia, by intercostal neuralgia, and is sometimes heard in an apparently normal chest. Such breathing, known also as cog-wheel respiration, becomes significant only when confined to the apex of a lung, for under these circumstances, especially if associated with dullness, it is one of the signs of incipient phthisis.

Divided respiration, an interval between the first and second acts, is found with bronchial breathing, and also in emphysema.

Prolonged expiration occurs in many cases of emphysema and of phthisis; the pitch remains low in the former, and rises in the latter. It must be remembered that expiration is prolonged and low-pitched in emphysema, and prolonged but high-pitched in phthisis.

Bronchial or tubular breathing, a description of which has been given, is heard over hepatized lung, as in pneumonia, over well-marked consolidation in

phthisis, and over compressed lung. From the filling up of the air-vesicles, sound-waves are no longer diverted into a multitude of lateral paths, as in the normal lung, and thereby almost dissipated, but extend, on the contrary, by successive reflections through the bronchial tubes, from the trachea to the surface, and thence to the ear; so that, instead of the diffuse, soft, breezy murmur of vesicular respiration, tubular breathing is heard over consolidated lung, with little if any loss of intensity, very much as sound-waves are reflected through a speaking-tube, or the air-pipes of a binaural stethoscope.

Broncho-vesicular respiration is found in disease where consolidation, or where compression of the lung, is not extensive enough to produce bronchial breathing, as in the early stage of phthisis, or when there is a small intrapleural effusion.

Cavernous breathing pertains chiefly to excavations in phthisis with easily collapsing walls; it is sometimes heard where the lung has not gone beyond consolidation.

Amphoric respiration occurs both in phthisis and pneumothorax; in phthisis over large cavities with tense walls, in pneumothorax if there be unobstructed bronchial communication.

## RECAPITULATION.

*Rhythm—Regular Succession of the Respiratory Acts.*

Interrupted rhythm: Slight deposit in lung.

Divided rhythm: Want of elasticity in lung.

Prolonged expiration: Want of elasticity in lung.

*Breathing.*

Vesicular: Uncomplicated lung.

Bronchial: Consolidated lung; compressed lung.

Broncho-vesicular: Moderate consolidation.

Broncho-vesicular: Moderate compression.

Cavernous: Flaccid cavity-walls.

Amphoric: Tense cavity-walls.

Exaggerated: Vicarious respiration.

Diminished: Plastic exudation, want of elasticity.

Absent: Fluid, air.

**Adventitious Signs.**—In addition to these changes of character in the normal respiration, which were shown to be an evidence of an altered condition, there are some new or adventitious sounds that serve a similar purpose.

There is but one way to become familiar with such sounds, and this is to embrace every opportunity of listening to them. But they are nowhere audible in a healthy person, and therefore, unlike the former

signs, can be studied only in the patient. Some preparation, however, is necessary for their recognition, and also for a correct interpretation of their significance.

Now, in view of the fact that all knowledge is more or less relative, an absolute grouping of these signs is not to be expected. Still, nearly every design will be fulfilled by the following classification, namely: harsh, sibilant, and sonorous breathing; crepitant, subcrepitant, bubbling, tracheal, gurgling, and splashing râles; metallic tinkle, and friction.

By harsh breathing is meant that the soft, breezy respiration has become rough through a variation in the bronchial mucous membrane that falls short of developing sibilant or sonorous breathing, but which, if carried a little further, would result in them. This kind of breathing is common in bronchitis, and especially in the chronic form of the malady.

Sibilant breathing, also termed sibilant râles, is a high-pitched, whistling sound that conveys the idea of dryness rather than of moisture. This noise is not constant, and can usually be heard during both acts of respiration, yet at times it is confined to inspiration.

Sonorous breathing, likewise called râles, is a low-pitched, dry, snoring sound of variable intensity. It

is present during both acts of respiration, is inconstant, and not infrequently is restricted to expiration.

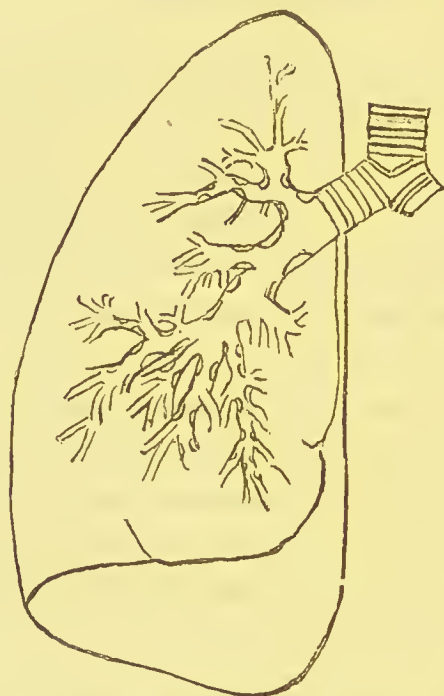


Diagram illustrating sibilant and sonorous râles.

Sibilant breathing has its origin in the smaller, sonorous in the larger bronchi, from a contraction in their caliber, either by spasm or pressure, or else by viscid secretions. The expiration is prolonged with both kinds of breathing. These sounds are heard in asthma, and in some cases of acute and in many cases of chronic bronchitis.



Crepitant râles are fine, dry, crackling sounds, an example of which may be obtained by rubbing the objective end of an adjusted stethoscope against a hairy chest. Râles of this nature are heard only during or toward the end of inspiration, and are superficial and rapidly evolved. They are frequently inaudible, because not developed unless the patient is made to breathe deeply. The sound probably originates in the pleura, for crepitation is not heard until the two surfaces of this membrane move one upon the other; it ceases when movement becomes impossible, and commonly returns with returning pulmonary mobility. Another explanation of the source of these râles is based upon the assumption that the walls of the air-cells are stuck together by a viscid secretion during expiration, and torn apart by each inspiration, which thereby causes the crepitation. These râles are nearly, if not quite, characteristic of pneumonia, yet certain conditions of the pleura, similar to what obtain in that affection, may give rise to just such crackling.

Subcrepitant râles are fine, bubbling sounds, generated in the smaller bronchi by air passing to and fro through fluid. They are distinguished from crepitant râles by their liquid quality, and by their occurrence with both acts of respiration. These râles are found

on each side of the chest, at the lower part of the lung, in capillary bronchitis, and in pulmonary œde-

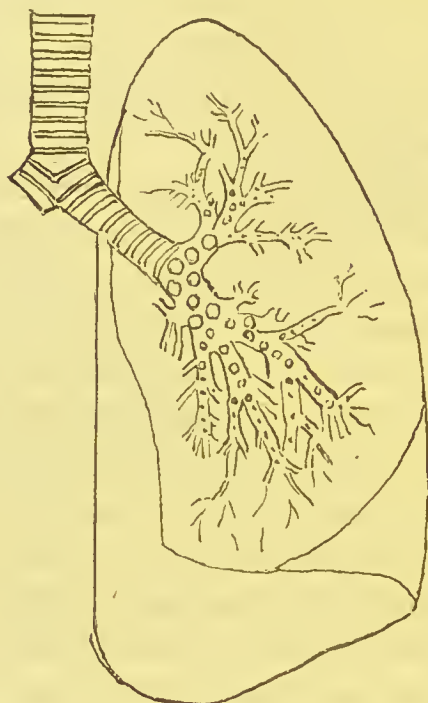


Diagram illustrating large and small bubbling râles.

ma. When limited to the apex, they may be one of the signs of phthisis. Lastly, they accompany resolving pneumonia, and sometimes follow an hæmoptysis.

Bubbling râles are developed by air passing backward and forward through fluid in the larger bronchi, and hence differ but in degree from subcrepitant râles.

Still coarser râles are produced after the same manner in the trachea, and from it take their name—tracheal râles. The bubbling râles are heard in bronchitis, in phthisis, and during the latter stage of pneumonia, while tracheal râles are peculiar to comatose and to moribund states.

Gurgling râles sound like the coarse, irregular, noisy flow of liquid from a bottle, and are the result of the passage of air through fluid into cavities. These râles occur in the excavations of phthisis and in bronchial dilatations, whose secretions impede the entrance of air. Loud clicks at intervals may be heard in connection with gurgles, and are supposed to be the bursting of bubbles in the vomicæ.

Splashing râles are sounds stirred up by shaking a patient's chest wherein there are both air and liquid. This operation, which is termed *succussion*, is performed with the ear directly upon the chest of the person examined, and a hand upon each shoulder. Such râles are practically restricted to hydropneumothorax; but it is within the range of possibility to mistake the splashing of fluid and air in the stomach for the signs of this disease.

Metallic tinkle, which is defined by its name, is a tinkling sound that is heard with irregular recurrence in hydropneumothorax, and more especially when

there is a bronchial communication ; and, while it is almost confined to this malady, it may possibly be audible in a large excavation of phthisis ; and, too, like the splashing râle, it can be transmitted from the stomach to the chest.

Friction is a grazing, rubbing, or crackling sound, according to the circumstances of its development. If the student, while listening, will slide the objective end of his stethoscope from side to side along the chest, he will find an example of one variety of this noise. It is commonly a to-and-fro rubbing, but it may be limited to inspiration. And there are times when this last is quite indistinguishable from the crepitation of pneumonia. Under these conditions, the history, symptoms, and other signs must determine its meaning, rather than a difference in the quality of the sound. Friction is heard in dry pleurisy, in the early stage of pleurisy with effusion, and near the beginning of pericarditis.

The rubbing returns after the reabsorption of fluid, when it may be styled secondary friction, and often remains a long period. A pericardial is distinguished from a pleuritic friction by the time and place in which it occurs. The first is synchronous with cardiac pulsation, the second with respiration ; pleuritic friction takes place in the pulmonary area,

usually at the side, pericardial friction in the region of the heart, often near its base. Now and then the action of the heart sets up an intrapleural friction, which is recognized as such with difficulty.

Finally, all extraneous sounds must be carefully eliminated during an examination. Among those most likely to happen are rubbing of the ear, the stethoscope, the clothes, and of fractured ribs, as well as the vibrations of moving bones and muscles, and also unusual noises carried to the ear through the chest from the throat or stomach.

#### RECAPITULATION OF ADVENTITIOUS SIGNS.

##### *Breathing.*

Harsh: Moderate thickening of bronchial mucous membrane.

##### *Râles.*

Sibilant: Contraction of small bronchi.

Sonorous: Contraction of large bronchi.

Crepitant: Slight exudation on pleura.

Subcrepitant: Air and fluid in small bronchi.

Bubbling: Air and fluid in large bronchi.

Tracheal: Air and fluid in trachea.

Gurgling: Air and fluid in excavation.

Clicks: Air and fluid in excavation.

Splashing: Air and fluid in pleural cavity.

Metallic tinkle: Air and fluid in pleural cavity.

*Friction.*

Exudation in pleura.

Exudation in pericardium.

**Vocal Resonance.**—The voice transmitted through the chest to the ear receives the name of vocal resonance; and there is a typical normal vocal resonance for different parts of the chest, just as a characteristic respiratory murmur was found. Generally speaking, this resonance is an ill-defined vibration, somewhat low in pitch, and of rather moderate intensity. But, as the listener approaches the larger bronchi, either in front or behind, the intensity increases, until over these tubes, and especially along the trachea, near the source of the vibration, the sound becomes unpleasantly loud, and closely borders upon articulation. As with fremitus, this resonance is greater upon the right than the left side, and just below the right clavicle than elsewhere. Likewise it is diminished by an overlying deposit of fat, and by a feeble or even high-pitched voice. Seeing that the low-pitched tones are transmitted with greatest velocity, it is not improbable that they are more distinctly audible for this reason. At all events, vocal resonance is best developed



by causing the patient to count one, two, three, in as deep a tone as possible.

Now, voeal resonance may be increased, diminished, or entirely suppressed by disease; and, like the respiration, it may assume a bronehial quality, which is termed bronehophony. Moreover, there are two modifications of this normal voeal resonance, called, respectively, amphorie voice and ægophony; the last is a tremulous, high-pitched sound, thought to resemble the bleating of a goat, the first a musical tone, such as vibrates with the voice in an empty flask.

When not only the voice, but also distinct articulation, is conveyed by way of the chest to the ear, it is known as pectoriloquy.

A similar transmission of whispered words is termed whispering pectoriloquy.

The voice, in a whisper, heard without well-defined articulation, is called cavernous whisper.

Increased voeal resonance is found, usually, over consolidated and over condensed lung-substance.

Diminished voeal resonance is the result of pleural effusion or of plastic exudation.

Absence of voeal resonance may be one of the signs of an intrapleural effusion, yet this vibration of the voice is not infrequently present even when the chest is quite full of fluid.

Bronchophony appears in pneumonia, and over vomicæ, and where the lung is compressed by fluid.

Amphoric voice, like amphoric breathing, is heard in hydropneumothorax, and over large excavations with tense walls in phthisis.

Ægophony is occasionally developed over compressed lung just below or at the level of a pleural effusion.

Pectoriloquy may take place under two conditions—one that of solidification, the other from excavation of lung-tissue. As a consequence of this it is evident that the assistance of other signs will be required to determine its meaning.

Whispering pectoriloquy occurs under like circumstances.

Cavernous whisper is one of the signs of an excavation.

Finally, all these signs depend upon a knowledge of local variations and a comparison of similar regions for their utility.

#### RECAPITULATION OF VOCAL RESONANCE.

Normal: Voice through normal chest.

Bronchophony: Voice through consolidation.

Amphoric: Voice in a cavity.

Ægophony: Voice in compressed lung.

Pectoriloquy: Articulate voice in cavity; in consolidation.

Whispering pectoriloquy: Whispered articulation in cavity; in consolidation.

Cavernous whisper: Ill-defined articulation in cavity.

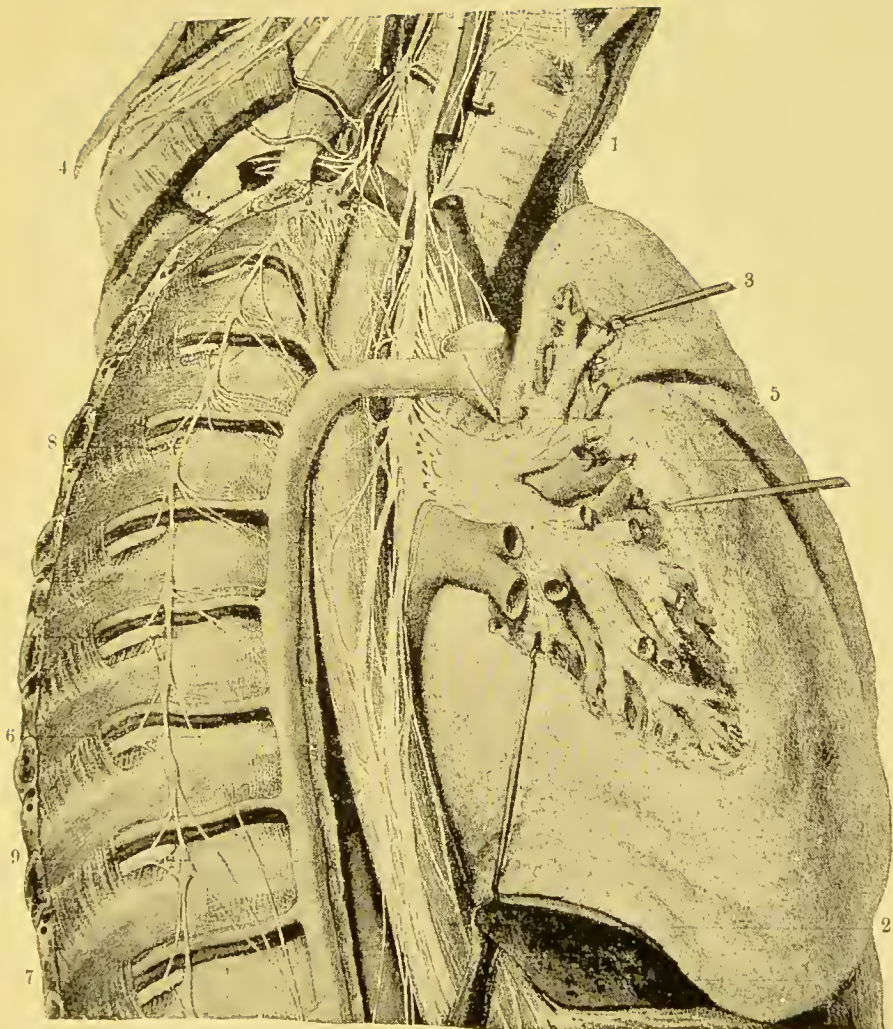
## DIAGNOSIS BY PHYSICAL SIGNS OF DISEASES OF THE LUNGS.

INASMUCH as actual disease varies often very much from the typical instances given in books, a consideration of the several physical signs, such as the previous pages have embraced, is of decided importance.

We found that an isolated sign may be of indefinite value, but joined with others it has a definite share in shaping diagnosis; that a sign may be normal in one and become abnormal in another place; and that most signs depend upon comparison for their significance. Hence a knowledge of the general principles of physical diagnosis is requisite for an intelligent study of thoracic affections.

### BRONCHITIS.

**Acute Catarrhal Bronchitis.**—In most instances acute bronchitis runs its course with no abnormal physical manifestations. This is due either to the



THORACIC CAVITY.—LUNG DRAWN FORWARD. BRONCHI AND PULMONARY VESSELS EXPOSED. VIEW FROM THE RIGHT SIDE (HIRSCHFELD).

1, trachea ; 2, oesophagus ; 3, arteria innominata ; 4, subclavian artery ; 5, superior vena cava ; 6, right azygos vein ; 7, thoracic aorta ; 8, right pneumogastric nerve ; 9, thoracic duct.





small extent of the lesion or the large size of the tubes involved. The diagnosis of such cases rests upon the history and symptoms, supported by the negative results of the physical exploration. Here we exclude pneumonia, pleurisy, and phthisis, by the absence of a combination of signs that might indicate one or more of these maladies. Even when the inflammation reaches the medium-sized tubes, in idiopathic bronchitis, there may be no physical signs; but, if present, the signs are a slight increase in the frequency of the breathing; and, as the affection advances, a thrill is sometimes communicated to the hand. There is little if any fever, 102° F. being about the average maximum elevation of temperature. On percussion, as a rule, there is no dullness. Early in the disease, upon auscultation, the intensity of the vesicular murmur is increased; later it is diminished, or wholly obscured by râles. An obstruction of a bronchus may cause a circumscribed suppression of the respiratory sounds, but the patient by coughing can usually effect their reappearance. The breathing, more especially in chronic bronchitis, may approach the character of sonorous—i. e., become what is termed harsh respiration—and thus continue throughout the disease; yet, now and then, there are sibilant and sonorous râles in the dry stage, with the addition

of large and small bubbling râles in the stage of secretion. These râles are variable in amount, heard over each lung, and may disappear, temporarily, from one or both sides. Expiration is prolonged. Vocal resonance is unaltered.

It is neither the intensity nor quality, nor yet the stability of the râles, so much as the amount of heart-failure and dyspnœa, that indicates how seriously the patient is affected.

**Chronic Catarrhal Bronchitis.**—The physical evidences of chronic are nearly those of acute bronchitis. We may have simply harsh respiration, or else the same lack of positive signs. Still, sibilant and sonorous breathing are more often present in chronic than the acute form of this disease. And when emphysema complicates chronic bronchitis, as it frequently does, the signs are modified by that affection. Then inspection shows more or less permanent expansion of the chest with deficient respiratory play, and at the same time considerable prominence of the auxiliary breathing muscles. On palpation the fremitus will be diminished, or at least unchanged. On percussion, instead of normal there may be vesiculo-tympanitic resonance. On auscultation the vesicular murmur is usually obscured by bronchial râles, and expiration prolonged; yet all respiratory sounds may be de-

creased and even absent from the lung wherein there is much emphysema. Vocal resonance is either unaltered or diminished. Fever is commonly absent.

A bronchitis that affects but one of the lungs is generally symptomatic; and, confined to the apex, it is very often one of the signs of phthisis. Finally, the duration rather than the character of the signs distinguishes chronic from acute bronchitis.

#### SUMMARY: SIGNS OF BRONCHITIS.

Inspection: Negative.

Palpation: Rhonchal fremitus.

Mensuration: Negative.

Calormetation: Moderate fever.

Percussion: Negative.

Auscultation: Sibilant and sonorous breathing; large and small bubbling râles.

**Bronchiectasis.** — As dilatation of a bronchus is sometimes found with chronic bronchitis, it must be distinguished from a similar expansion occurring in phthisis with pulmonary excavation. Now, simple dilatation gives rise to the auscultatory signs of a cavity; but, on the other hand, there is seldom much abnormal dullness, and though there may be some dullness from an accumulated secretion, and possibly

from compression of the adjacent lung, yet it does not precede the signs of excavation as in phthisis, and it is perceptibly diminished by evacuating the contents of the bronchial cavity. Moreover, in the last stage of phthisis dullness generally surrounds the vomieæ, whereas in dilated bronchi the resonance around the cavity is frequently normal. Then, too, the physical signs are almost stationary in the latter affection, while in phthisis they are always or nearly always progressive.

Fever is absent during a greater part of the time with dilatation of the bronchi, but with phthisis it is present through most of the progress of the disease.

Furthermore, the disproportion in bronchiectasis between the general condition of the patient and the local manifestations is very noticeable; and, lastly, a careful inquiry into the history and symptoms will lead to a correct interpretation of the existing physical signs.

**Acute Capillary Bronchitis.**—When the inflammation extends to the smaller bronchi or originates in them, either of which is very liable to happen in children and in old persons, there are additional signs of bronchitis to those already given.

It is seen by inspection that breathing is rapid,

that all the auxiliary muscles are brought into use, and that, difficult in any attitude, it is quite impossible to breathe while lying down. Moreover, with obstruction enough to cause an extensive collapse of the lobules, instead of the customary expansion of the chest, there will be a falling in at each inspiratory effort. The face is flushed, or in extreme cases livid and bedewed with sweat, and the lips and finger-tips are cyanotic.

Palpation detects an increase in the labored respiratory acts to forty, sixty, and even eighty in the minute.

The temperature of capillary bronchitis ranges from 101° to 103° F., and the fever often subsides with the progress of the malady.

Upon percussion the resonance is normal or may be vesiculo-tympanic, with possibly some dullness at the lower portion of the lung behind. Where the upper lobes become emphysematous, the resonance there is vesiculo-tympanic. Where the lower lobes are collapsed or consolidated, there the percussion note is dull. But wherever neither emphysema nor collapse nor yet consolidation exists, the resonance is normal.

Upon auscultation the vesicular murmur may be diminished or quite absent. It is sometimes dimin-

ished and at other times absent where there is vesicular emphysema. It is absent from the plugging of a bronchus. It is always somewhat diminished by the disease and frequently smothered by the râles. When the affection is general, sibilant breathing may be heard throughout the chest, accompanied or followed by subcrepitant râles, which are bilateral. These subcrepitant râles are present in every case of capillary bronchitis, and they not uncommonly mask the auscultatory signs of a complicating lobular pneumonia.

SUMMARY: SIGNS OF CAPILLARY BRONCHITIS.

Inspection: Orthopnœa, dyspnœa, cyanosis.

Palpation: Respiration forty to eighty.

Mensuration: Negative.

Calormetation: Temperature, 101° to 103·5° F.

Percussion: Normal, vesiculo-tympanitic.

Auscultation: Sibilant, subcrepitant râles.

**Croupous Bronchitis.**—The physical evidences of croupous or fibrous bronchitis chiefly depend upon the presence of membranes, which are formed in the bronchi. This variety of bronchitis is exceedingly rare, yet all students that have once seen the peculiar sputa will have no difficulty in recognizing the disease.



Just previous to the expulsion of a cast there is usually some dyspnœa. The vesicular murmur is diminished and even suppressed over a portion of the lung when a large bronchus is obstructed. In some instances a rustling or flapping sound is heard, together with the râles of catarrhal bronchitis, should this coexist.

There may be dullness, from a temporary collapse of lung-substance.

Fever accompanies acute but not chronic forms of this disease.

However, it is upon the appearance of the branch-like or cylindrical tube-casts, which were described under the head of sputa, that the diagnosis of croupous bronchitis rests.

All kinds of bronchitis are distinguished from pneumonia and from phthisis mainly by the absence of dullness; and if associated with these affections it is diagnosticated by the presence of bronchial râles in addition to the signs of the other malady.

#### ASTHMA.

Spasms of the bronchi occur primarily or follow emphysema or else bronchitis. The physical signs of asthma are those developed during a paroxysm.

It is seen upon inspection that the respiration is slow and laborious, with dyspnœa marked in expiration.

On palpation, vocal fremitus is normal. On percussion, resonance is either normal or vesiculo-tympanic.

On auscultation, sibilant and sonorous breathing are revealed, widely diffused throughout the chest, of decided intensity, and chiefly expiratory. Moreover, considerable wheezing is audible at a distance from the patient, and with the subsidence of the spasm bubbling râles are heard; but when the paroxysm is secondary to bronchitis, it is not unusual for these râles to exist from the commencement. The vesicular murmur, which is jerky in rhythm, may have its intensity increased or diminished. Vocal resonance is not changed. The temperature is unaltered.

With the relaxation of idiopathic bronchial spasm there is a return of the normal respiratory murmur; but in symptomatic asthma the signs of emphysema or bronchitis continue and become more distinct.

There is aphonia in laryngitis, with stridor rather than wheezing, and the dyspnœa is mainly inspiratory. On the other hand, there is neither aphonia nor stridor in asthma, but wheezing and expiratory

dyspnœa. While in œdema of the glottis there is no aphonia, yet, as in laryngitis, the dyspnœa is inspiratory; and, above all, though present in asthma, sibilant and sonorous breathing are absent both in œdema of the glottis and laryngitis.

#### SUMMARY: SIGNS OF ASTHMA.

Inspection: Respiration slow, labored, gasping.

Palpation: Rhonchal fremitus.

Mensuration: Negative.

Calormetation: No fever.

Percussion: Resonance normal, vesiculo-tympanic.

Auscultation: Sibilant, sonorous breathing, wheezing.

#### HÆMOPTYSIS.

For the diagnosis of bronchorrhagia we chiefly depend upon inspection. Hence, should a patient expectorate bright-red, frothy blood, in all probability it would come from the bronchi, providing there is neither buccal nor naso-pharyngeal hæmorrhage. The trickling of blood from the pharynx into the throat, whence expelled by a cough, now and then proves a cause of alarm to the patient, and of mistake to the physician.

While it is true that hæmoptysis is usually fol-

lowed by large and small bubbling râles in the involved region, nevertheless physical exploration, at this time, should be performed gingerly, if at all, for the risks attending a disturbance of the patient are greater than the value of the acquired information. But later, when an examination can be made with safety, it is well, if possible, to find the source of the bleeding.

Although pulmonary hæmorrhage, so called, is commonly due to phthisis, heart disease, and to aneurism, with a frequency in the order enumerated, yet the flow of blood may be bronchial in its origin, and bronchial only. But, still, phthisis, or a predisposition to phthisis, is most often the condition in which there is spitting of blood, and this may precede all other appreciable signs. With a copious hæmorrhage the temperature usually declines.

Finally, if the bleeding takes place in the stomach, the blood thrown up can be distinguished from that which comes from the bronchi by its color, consistency, and mode of exit. It is dark, mixed with food, and vomited; whereas blood from the lung is light red, frothy, without ingesta, and wells from the throat with scarcely a cough.

## PULMONARY EMPHYSEMA.

In emphysema both lungs are involved, as a rule, but not to a like extent. The upper border of each lung may be affected, or the upper in one and the lower border in the other.

There is a senile emphysema in which contraction rather than expansion of the lung takes place. Here, too, a dilatation of the air-cell occurs, yet the whole lung is decreased in bulk from an atrophy of the pulmonary tissue.

By inspection, in emphysema, the breathing is seen to be labored and prolonged in expiration. There is a drooping of the shoulders; the muscles of the neck stand out prominently; and the contour of the chest is more or less altered, by an expansion just below the clavicles, or below the clavicle of one side and the nipple of the other. Less commonly there is an arching forward of the sternum, an antero-posterior curvature of the dorsal spine, and, in fine, a general expansion of the thorax that is suggestive of a barrel, and which is therefore appropriately termed barrel-shape.

On the other hand, in senile emphysema, there is neither this general expansion nor any local bulging of the thorax.

The only visible cardiac impulse in the first-mentioned variety of this malady is confined to the epigastric region.

Upon mensuration it may be found that the circumference of the lower part of the chest becomes even less during inspiration, while, higher, there is practically no expansion. In fact, the chest-wall seems to be lifted up bodily, rather than expanded, and breathing is almost wholly diaphragmatic.

On palpation, though vocal fremitus is often unaltered, it may be either diminished or absent, while in senile emphysema the fremitus is not infrequently exaggerated.

By percussion, in distinctive cases, the quality of the note is vesiculo-tympanic, yet in many instances it is simply pulmonary, or but slightly intensified resonance.

A marked distention of one side may render the other dull by comparison, when in reality there is no dullness.

The area of cardiac flatness and dullness disappears with great expansion of the lung, and this organ may extend so low as to obliterate the dullness as well as flatness of the liver.

Finally, there is no difference, in emphysema, between resonance on full expiration and that on full



inspiration, and this is shown by respiratory percussion.

Upon auscultation over the emphysematous portion of the lung, the breathing is diminished or suppressed, or else, what is more common, inspiration is either deferred or absent, and expiration is prolonged and of low pitch. Thus the natural ratio of the two respiratory acts becomes inverted; nay, the expiratory may be many times the length of the inspiratory act. Also, fine bronchial râles are heard where bronchitis coexists.

Vocal resonance, in this affection, is not to be relied upon, for it may be absent, or normal, or even increased.

The sounds of the heart are partially or wholly obscured in all but the tricuspid area.

As the disease is unattended by fever, a rise in the temperature would indicate some intercurrent affection.

The physical signs of emphysema could be mistaken only for those of pneumothorax.

Now, the resonance in the latter is always tympanic, and, moreover, one-sided, whereas it is seldom if ever purely tympanitic in the former, but rather vesiculo-tympanitic, and on both sides. Furthermore, the respiration, if heard, in pneumothorax is amphoric, in emphysema not; and, too, the difference

in the history and symptoms of the two maladies is very clearly defined.

SUMMARY: SIGNS OF EMPHYSEMA.

Inspection: Breathing slow and labored.

Palpation: Fremitus diminished; unaltered.

Mensuration: Slight if any expansion.

Calorimetation: No fever.

Percussion: Resonance normal; vesiculo-tympanitic.

Auscultation: Respiration diminished; absent; or, prolonged low-pitched expiration.

PULMONARY ŒDEMA.

In pulmonary œdema the serum is usually diffused throughout the lung, but, when the fluid is abundant, much of it gravitates to the dependent parts of the organ.

By inspection, the frequency of the respiration is found to be increased, and with this there is considerable dyspnœa.

On palpation, the vocal fremitus in one instance may be exaggerated, and in another diminished; therefore, it is of little positive value in the diagnosis of this affection.

Upon percussion, with a great deal of œdema,

there is dullness, and it is most noticeable behind, at the lower portion of the lung.

Through auscultation it is learned that the vesicular murmur is either absent, or at least quite feeble. And subcrepitant râles, often very fine, are heard over the involved region. Vocal resonance is no more reliable than vocal fremitus. There is no fever attending this condition.

From capillary bronchitis, pulmonary œdema differs in having slight dullness and no fever. And the absence of bronchial breathing, of fever, and of well-marked dullness would exclude inflammation of the lungs. Moreover, œdema is bilateral, while, as a rule, pneumonia is unilateral. The sputa of the former are copious, frothy, and watery ; of the latter scanty, viscid, and of a characteristic color. Lastly, it is a help to know that œdema of the lungs is commonly associated with dropsy in the body elsewhere.

#### SUMMARY : SIGNS OF ŒDEMA.

Inspection : Rapid, difficult breathing.

Palpation : Fremitus diminished.

Mensuration : Negative.

Calormetation : No fever.

Percussion : Some dullness.

Auscultation : Bilateral subcrepitant râles.

## PNEUMONIA.

An inflammation of the pulmonary tissue does not necessarily involve an entire lobe, and scarcely ever the whole lung. It begins at some one spot, usually the lower lobe, and thence rapidly extends, oftentimes irrespective of the intervening fissure.

The malady is termed single or double pneumonia, as one or both lungs are affected; and when the inflammation fails to reach the surface of the lung, it is known as central pneumonia.

For convenience, the disease is divided into three stages, each with its special signs. One part, however, may have arrived at the second, while another is progressing toward this stage. And now and then, owing to a speedy hepatization, the first appreciable evidences of pneumonia are those of the second stage—consolidation.

The three stages, with respect to the physical signs, are congestion, solidification, and resolution.

*First Stage.—Congestion.*

*Inspection.*—In the first stage of pneumonia there is seen rapid breathing, of a panting nature, and, withal, some restriction in the respiratory play of the affected side. A view also of the sputum is of assist-

ance, for, with a limited central pneumonia, this and the fever may be the only physical manifestations of the disease. First, the patient spits a frothy mucus; then, less easily, a sticky, translucent material, which adheres to the receptacle. The color of the sputum is amber, orange, or brick-dust, according to the amount of blood with which it is tinged, for blood is diffused through the expectoration, and not usually in streaks. Such matter is called *rusty sputa*. Sometimes the material coughed up is quite fluid, and shades into a prune-juice color, which is considered a bad omen. Children, it must be remembered, have pneumonia often without expectoration.

*Palpation.*—By counting the respirations, they are found to have increased to forty, and even eighty, a minute; and, while there is commonly an exaggeration of vocal fremitus on the affected side, in some instances this is not perceptible.

*Calormetation.*—The temperature rises quickly to 103°, 104°, or 105°, and even to 106° F., and there remains for nearly twenty-four hours, after which there is a daily morning remission, followed by an evening exacerbation. As a fair elevation, 104° F. might be taken, and a moderately high fever seems to be rather favorable than otherwise. At all events, the student is cautioned against too much alarm at

very temporary flights of the index, and is likewise reminded of the proneness of children to high temperatures. On the other hand, old persons may be in most critical conditions of pneumonia with no fever.

*Percussion.*—Over the involved area there is a rise in pitch toward the end of this stage, which amounts to moderate dullness, providing the disease extends to the surface of the lung.

*Auscultation.*—In the affected portion of the lung the intensity of the vesicular murmur varies. It may be exaggerated, but it is often diminished.

A multitude of crepitant râles are soon heard, which are fine, crackling sounds, developed at or near the close of an inspiration. But, unless the patient is made to breathe deeply, these râles will be overlooked; and when consolidation takes place rapidly, they are not produced. Vocal resonance is increased.

*Second Stage.—Solidification.*

*Inspection.*—The respiratory action of the chest-wall is now chiefly confined to the unaffected side, where it is magnified and still rapid. Actual dyspnoea, however, is not always present.

*Palpation.*—Over the affected portion of the lung, in most instances, vocal fremitus is increased.



But in some there is either no alteration or the vibration is actually diminished.

*Calormetation.*—There is a continuation of the high thermometrical range, and while the highest

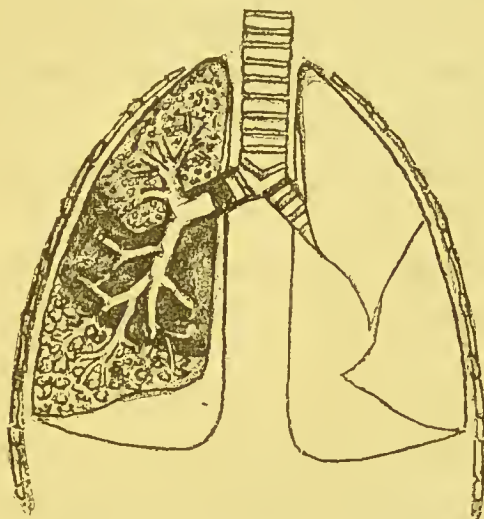


Diagram showing, from above downward, the three stages, congestion, solidification, and resolution of pneumonia.

point may be reached early in this stage, sometimes the fever is at its maximum elevation just before a crisis, which is commonly later.

*Percussion.*—Well-defined dullness over the area of consolidation usually marks this stage, while over non-hepatized lung the resonance is intensified, or possibly vesiculo-tympanitic.

But percussion signs depend upon the situation

and the extent of the solidification, for central pneumonia may afford only slight dullness, or none whatever; whereas, if the whole lung is involved, there will be flatness rather than dullness. Moreover, there may be tympanitic resonance either from a close proximity of the consolidation to the stomach, distended by gas, or because of the nearness of the pneumonia to a large bronchus.

Now, between the tympanitic resonance of pneumonia and that of pneumothorax there is this difference, namely, an unmistakable feeling of resistance in the former, which is not felt in the latter, upon percussion.

In some instances, furthermore, a cracked-pot sound can be detected in pneumonia, and these are mainly when percussing over a large bronchus, whence the air is driven as from an excavation.

*Auscultation.*—Where consolidation is, there, as a rule, bronchial breathing is heard, and likewise bronchophony, and frequently whispering pectoriloquy.

Still, these signs may possibly fail, from an overlying pleuritic exudation or from an obstruction to a large bronchus. In the latter instance the breathing may be restored by a series of forced coughs.

But pneumonia occurs at times with some dull-

ness over a circumscribed area where there is no bronchial breathing; not, in this case, from plugging of a bronchus, but on account of the feebleness of the respiration. Here a forced cough will almost invariably develop a bronchial puff, yet the bronchial breathing is not permanently restored.

The peculiar vibration of the voice, known as bronchophony, also may be developed where bronchial breathing can not, and thus prove a very useful sign.

Finally, upon the unaffected side of the chest there is usually an exaggerated or puerile respiration.

### *Third Stage.—Resolution.*

There are no physical signs by which gray can be distinguished from red hepatization; hence, we pass to the manifestations of resolution.

The clearing up takes place step by step, and its progress is attended by physical signs of the receding deposit.

*Inspection.*—With resolution the normal respiratory action of the chest is resumed, and the sputa become yellow in color and abundant.

*Palpation.*—Vocal fremitus is lessened in intensity.

*Calormetation.*—The temperature returns to the

normal, or falls to the subnormal, either suddenly or by degrees—more often, however, in the former manner. And it will be observed that the panting respiration terminates with the decline of the fever, before resolution has made much progress. Thus rapid breathing seems to depend not upon the extent of the consolidation alone.

*Percussion.*—The decrease of dullness is quite gradual, and a slight amount remains a long time.

*Auscultation.*—From bronchial breathing there is a return to broncho-vesicular, and thence to pulmonary respiration, with the disappearance of bronchophony and of increased vocal resonance. And associated with these changes crepitant and subcrepitant râles are heard, the latter of which predominate. Moreover, there may be large, bubbling râles as well.

The physical signs of pneumonia in the very old are often obscure, because of the rigidity of the chest-wall and the senile changes in the lung itself. Fever, too, is either slight or absent, and there may be no dyspnoea, not much acceleration of the breathing, and, what is more, little if any expectoration. In fact, bronchophony, or else the bronchial puff before mentioned, may be the only physical manifestation of the disease.

**Purulent Infiltration.**—When, instead of resolution,

purulent infiltration occurs, the temperature remains high, dullness and bronchial breathing continue, and, in addition, high-pitched, gurgling râles are heard, while the expectoration becomes both profuse and purulent.

**Abscess.**—A pulmonary abscess can be detected, after a bronchial communication has been established, by the physical signs of an excavation, which are given in connection with those of phthisis.

**Lobular Pneumonia.**—The physical signs of lobular are mainly those of lobar pneumonia, but, as a rule, they are confined to small areas; and, except a large number of contiguous lobules are affected, these signs are obscured by the physical manifestations of the bronchitis, which the pneumonia usually complicates. For this reason fine subcrepitant râles are more often heard than crepitant râles, and dullness is not easily made out.

In this disease the temperature rises less suddenly than in lobar pneumonia, is more irregular in its course, and more gradual in its decline.

#### SUMMARY: SIGNS OF PNEUMONIA.

Inspection: Panting, rapid respiration; rusty sputa.  
Palpation: Breathing forty to eighty; fremitus increased.

Mensuration : Negative.

Calormetation : Temperature 103° to 105° F.

Percussion : Dullness—1st, slight ; 2d, complete ; 3d, decreasing.

Auscultation : 1st, crepitant râles ; 2d, bronchial breathing and bronchophony ; 3d, broncho-vesicular breathing, crepitant, subcrepitant, and bubbling râles.

#### PLEURISY.

The products of an inflammation of the pleura are fibrin, serum, and pus ; now fibrin, then serum with fibrin, and then again fibrin, serum, and pus. Moreover, there is a resulting formation of new connective tissue with adhesions or at least thickening of the pleura.

As a rule, these inflammations are confined to one side of the chest.

Where there is an exudation of fibrin alone, it is commonly circumscribed, and the disease is called dry pleurisy ; and if the exudation is followed by a large effusion of serum, the disease is termed sub-acute pleurisy. But when the exudation of fibrin is attended by an effusion of pus as well as serum, then the malady is known as empyema.



Hence it follows that there are three phases of the inflammation to consider ; namely, acute pleurisy, subacute pleurisy, and empyema.

The signs of a collection of serum embrace those of purulent effusion, and there is but one method by which the nature of the fluid can surely be determined, to wit, the withdrawal of a specimen for inspection. Therefore, we shall study the physical evidences of fibrinous exudation, fluid effusion, absorption, and adhesion, in the order named, and dwell, in passing, upon the manner of drawing out some of the contents of the pleural cavity.

**Fibrinous Exudation.**—The signs of acute pleurisy that are seen upon inspection are mainly those caused by the all-absorbing pain, and this is usually located at the lower part of the affected side where there is greatest pulmonary mobility. The patient is found doubled over toward this side, which he grasps to stay the movement of the ribs and thus lessen his suffering. He breathes with difficulty in a jerky, catching, rapid manner and superficially, for a deep breath fills out the lung and thereby intensifies his pain. Moreover, from a like motive he endeavors to suppress his cough, which is short, dry, and extremely painful ; consequently, the customary respiratory action of the diseased side is considerably restricted.

With the advance of the malady a friction vibration may be perceptible on palpation, while at the same time the vocal fremitus decreases.

An irregular range of temperature is shown by calormetation that rarely exceeds 103° F., seldom reaches 101° F., and often falls short of the fever-mark.

On percussion, the resonance rises in pitch with the exudation of fibrin to well-defined dullness where there is very much plastic material.

On auscultation, the respiratory murmur is both weak in intensity and jerky in rhythm; but the most prominent sign that forces itself upon the ear is one of the various forms of friction; and while this friction may accompany either the first or second or each respiratory act, it is more often audible with inspiration and at the lower part of the chest, where the excursion of the lung is most extensive. Further, for just this reason, friction now and then escapes observation as the sufferer seeks to control the movements of the lung in order to keep the pleural surfaces apart. Hence, if the suspected friction be inaudible, it is a good plan to ask the patient to take a deep breath or to cough, the latter of which will be followed of necessity by the desired inspiration.

To distinguish acute pleurisy from the first stage

of pneumonia, we keep in mind the low range of temperature of the former as compared with the high range of the latter, the friction of pleurisy instead of the crepitant râle of pneumonia, and the characteristic sputa of the last-named in place of no expectoration in the first-named affection.

There are two conditions that closely simulate dry pleurisy, namely, neuralgia of the intercostal nerves, and rheumatism of the intercostal muscles. In neuralgia there are some painful points along the course of the nerve, while in rheumatism there is wide-spread tenderness. Moreover, pressure aggravates the tenderness of the one and alleviates the pain of the other, whereas in pleurisy both superficial pain and tenderness are less common and less affected by external pressure. Furthermore, though fever may be associated with neuralgia and with pleurodynia, yet it oftener accompanies inflammation of the pleura, and, above all, friction appears in pleurisy, while it is never heard as a result of intercostal neuralgia nor of rheumatism.

**Serous Effusion.**—As fluid collects in the chest the opposing surfaces of the pleura are gradually separated, and, if the effusion is sufficiently extensive, friction disappears, to return, however, with the reabsorption of the liquid; consequently, with

these altered conditions there is a corresponding change in the physical manifestations.

*Inspection.*—Our attention is drawn, not only to the lack of motion upon the affected side, but also, if the effusion is abundant, to the enlargement of this side, and at the same time to the increased respiratory play of the unaffected portion of the chest. Still, this alteration in the size of the chest is not so common in adults as in children, for the elasticity of the thorax diminishes with advancing years.

While the breathing is somewhat increased in frequency by an effusion, it is neither the panting seen in pneumonia nor the dyspnoea that attends acute pleurisy. In fact, the patient is usually not conscious of much difficulty with the breath, except upon exertion. By closer inspection we may find that the intercostal spaces bulge, or at least remain stationary, with inspiration.

Where a large effusion occupies the right pleural cavity, the heart is displaced to the left, and, contrariwise, the organ is pushed to the right by an effusion in the left pleural cavity.

*Palpation.*—The position and force of the cardiac impulse, however, are better determined by the hand than by the eye, and likewise the frequency of the

respiration, which varies with an effusion from twenty to thirty a minute.

Vocal fremitus disappears in the region of the fluid, and, on the other hand, is sometimes exaggerated above the effusion, over the compressed lung.

This absence of fremitus where there is fluid is a very constant rule to which there are but few exceptions.

*Mensuration.*—Though upon forced expiration the semi-circumference of the affected side often measures from one half to two and even three inches more than the other, yet quite an amount of fluid may gather at the expense of the viscera, without perceptible enlargement of the thorax.

*Calormetation.*—While the thermometer may indicate 100°, 101°, or 102° F., and even a greater elevation of temperature, still, in quite a number of instances, there will be no fever.

*Percussion.*—From below up, as far as the fluid extends, there is flatness, but a change in the level of this flatness, by altering the patient's position, is not so common in pleuritis as with passive effusions.

The line of flatness takes something of a curve, with its lowest point near the spinal column and its highest in the axillary line, whence it descends toward the sternum. Nevertheless, adhesions modify

this line, and large effusions quite obliterate the curve.

In addition to the flat sound, there is a sense of resistance communicated to the hand, in percussing, which is very significant of the presence of fluid.

As we pass from the effusion to the compressed lung, the flatness changes to dullness; and this transition is best appreciated after causing the patient to take several deep inspirations. But in some few instances the resonance is vesiculo-tympanitic instead of dull over the displaced lung, and the unaffected side is thereby rendered dull by comparison. Now, this vesiculo-tympanitic resonance, which occurs above an effusion, is thought by some writers to depend upon dilatation of the air-cells from a permanent expansion of the chest. It seems quite as probable, however, that by a certain pressure of fluid, just short of producing collapse, the volume of the lung is contracted, so that the air of the bronchi, surrounded by this partly apneumatized lung, gives a sound on percussion that results in a modified tympanitic or, in other words, vesiculo-tympanitic resonance; and when the pressure passes beyond this limit, the air-vesicles and possibly the bronchi collapse, and then well-marked dullness is developed.

*Auscultation.*—The auscultatory signs of an effu-



sion depend upon the acoustic law that "sound-waves are transferred from air to liquids or to solid bodies

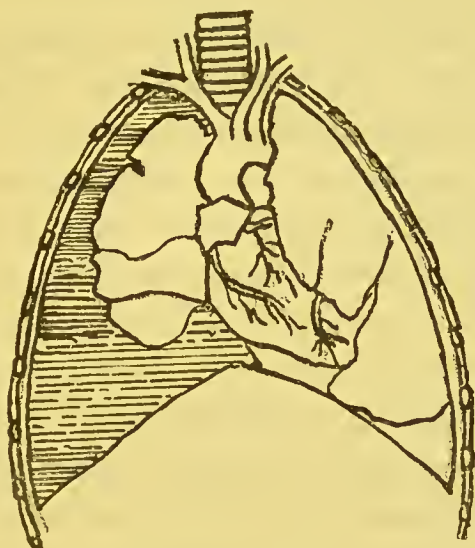


Diagram showing the right pleural cavity filled with fluid.

with great difficulty." In consequence of this, the respiratory murmur is either inaudible or, at most, very faintly heard through the fluid; and the sound, if perceptible, is that of muffled bronchial rather than vesicular respiration. This is best appreciated by listening to both sides of the chest; and, indeed, comparison is very essential in all methods of physical exploration.

With children, bronchial breathing is heard through fluid somewhat more clearly than with

grown persons; and, too, there are examples even in adults where bronchial respiration can be distinctly heard; but this does not invalidate the rule, for the lung under these circumstances is pressed toward the back, and there bound by adhesions. When such is the case, let the examiner carry his ear in the direction of the axillary line, and the sound almost invariably decreases and finally becomes lost upon reaching this locality. The axillary line is a most important point in which to percuss as well as listen in many thoracic diseases.

Vocal resonance, though absent below the level of the fluid in a number of instances, is not infrequently present, and present, too, where vocal fremitus is suppressed. Furthermore, at or near the level of the effusion a bleating sound, *ægophony*, is now and then audible; while above the fluid, in the compressed lung, there is either broncho-vesicular, bronchial, or else, possibly, cavernous breathing; and upon the unaffected side there may be, owing to vicarious work, what is called puerile respiration.

It is necessary to decide between a collection of fluid caused by pleuritis and one the result of a passive effusion.

In the first place, the history and symptoms are different. There is, for instance, no pain attending

the latter, and it is commonly associated with dropsy in other parts of the body. The hydrothorax has for its remote cause an organic lesion of the heart or the kidneys, and, as a rule, is bilateral; whereas it is the exception for pleurisy to occur in both sides. A passive effusion is not preceded by friction; and, moreover, fever is always absent. Lastly, the fluid of hydrothorax more easily changes its position with the movements of the patient than that of pleurisy, and thereby alters the level of flatness.

To distinguish an effusion of pleurisy from the solidification of pneumonia, we must likewise consider the history and symptoms, as well as the physical signs.

The invasion of pneumonia is signalized by a decided rigor; that of pleurisy by chilliness, without much shivering. Pain may or may not be prominent in both affections.

An effusion usually gravitates to the lower part of the chest; consolidation may take place at any point in the lung.

In pleurisy there is immobility of the affected side, with expansion; in pneumonia there is some immobility, but no expansion.

The sputa of the last-named are characteristic, those of the first-named affection not.

Fremitus is absent in pleurisy, while in pneumonia it is not only present but exaggerated.

The respirations are from forty to eighty in the one, and from twenty to thirty a minute in the other.

The half circumference is increased by an effusion and not modified by consolidation.

The temperature ranges from  $100^{\circ}$  to  $103^{\circ}$  F. in pleurisy, while in pneumonia it commonly lies between  $102^{\circ}$  and  $105^{\circ}$  F.

Where there is fluid there is flatness; hepatization gives dullness.

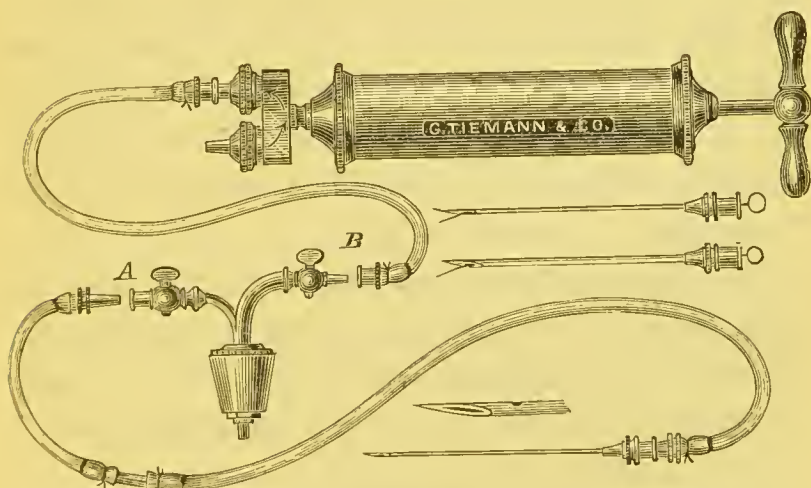
Upon auscultation vocal resonance and the vesicular murmur are absent in pleurisy, but in pneumonia both bronchial breathing and bronchophony are heard.

*Purulent Effusion.* — An inflammation of the plenra may be followed by a pouring out of pus at once, or after a time the serum may be changed to pus. Such a condition is known as empyema, and also as pyothorax.

Now, if there is fluid in the pleural cavity, the physical signs will be the same, whether it is pus, blood, or serum. Therefore, the student has but to turn back to "Fluid Effusion" for the physical manifestations of this disease.

With signs of an effusion in the pleural cavity,

there is good reason for suspecting pus, should repeated rigors and sweats occur, as well as a higher range of temperature than ordinarily obtains in sub-acute pleurisy. But positive evidence of the nature of the effusion can be gained only by drawing out some of the fluid for inspection. And though this may be accomplished by means of a hypodermatic syringe, yet an aspirator is preferable, for the ex-



Aspirator.

hausting power of the latter instrument is much the stronger, and the caliber of its needles better suited to the purpose.

A good position for the patient during this operation is to sit sidewise upon a chair, and a convenient attitude for the operator is to kneel upon the

floor. The place where the needle should be introduced is either the fifth, sixth, or seventh intercostal space, just to the outer side of the scapular line. Let the operator for a moment firmly press his thumb into the space selected, and then insert the needle where he has made the depression. The entrance should be effected nearer the lower than the upper part of the intercostal space, and the instrument thrust not deeper than one inch, to escape the intercostal vessels in the first, and to avoid reaching the lung, or possibly the diaphragm, in the second instance. If the needle is pushed through the fluid into the lung, not only is that organ wounded, but the object of the procedure defeated.

And, with all these precautions, in view of a possible injury to the expanding lung, it is perhaps advisable to use a small trocar and cannula, now made to fit the aspirator, rather than the sharp needle which usually accompanies that instrument.

**Absorption.**—As the fluid recedes, the fremitus slowly returns, flatness disappears from above downward to the lower part of the chest, and vesicular respiration is again heard, but in diminished intensity. With this comes a return of the friction, which often lasts for a considerable time. And, too, at the lower part of the chest, because of the thick-



ened pleura, flatness may continue for an indefinite period.

A contraction of the chest does not usually follow acute pleurisy, but it frequently succeeds the subacute form, when, as shown by mensuration, there is a decrease in the semi-circumference of the affected side, toward which the heart is then drawn.

It is now and then a difficult matter for many examiners to determine whether flatness, in a given case, is due to fluid or to the remains of an old pleurisy; but, in the latter instance, if the half-circumference is not decreased, it certainly will not be increased. Moreover, the heart will be drawn toward, rather than pushed from, the affected side; and, furthermore, the breathing will be indistinctly heard through the area of flatness, upon very careful and repeated auscultatory exploration; but, however faint, it will be vesicular, instead of muffled bronchial respiration.

**Adhesion.**—With the absorption of a sero-purulent effusion, there may be an adhesion of the costal and pulmonary pleura, and attachments may also follow a plastic exudation, or form in the very commencement of the disease. Under these circumstances there is considerable restriction of the respiratory play of the affected side; the half-circumference is dimin-

ished, the heart is drawn toward the contraction, vocal fremitus is either decreased or absent, and there is dullness or flatness, in proportion to the amount of new connective tissue. Lastly, the respiratory murmur is suppressed, or, at least, feeble; while here and there friction-sounds remain audible.

SUMMARY: SIGNS OF SUBACUTE PLEURISY.

Inspection: Marked immobility with expansion; no distinctive sputa.

Palpation: Vocal fremitus absent; breathing twenty to forty.

Mensuration: Increased semi-circumference.

Calormetation: Temperature  $100^{\circ}$  to  $103^{\circ}$  F.

Percussion: Flatness.

Auscultation: Friction; absence of vesicular murmur and vocal resonance.

Contrasted with signs of pneumonia:

Inspection: Slight immobility with no expansion; rapid, panting respiration; characteristic sputa.

Palpation: Vocal fremitus increased, breathing forty to eighty.

Mensuration: Negative.

Calormetation: Temperature  $102^{\circ}$  to  $105^{\circ}$  F.

Percussion: Dullness.

Auscultation : Crepitant râles ; bronchial breathing and bronchophony.

## HYDROTHORAX.

A passive effusion in the chest, termed hydrothorax, is detected by the same physical methods that are employed to demonstrate the fluid of pleurisy.

In the latter, however, the effusion is commonly single, whereas in the former condition it is always double. But the dropsy of the two sides is not equally extensive ; and, unless the chest is full, the position of the liquid shifts with the movements of the patient more easily than in pleurisy. With fluid on one side, the lateral displacement of the heart is much greater than when both sides are affected. Moreover, there is no fever in hydrothorax. The effusion is not preceded by friction ; it is commonly associated with dropsy elsewhere, and is frequently the result of disease of the heart or kidneys.

## PNEUMOTHORAX — PNEUMO-HYDROTHORAX — PNEUMO-PYOTHORAX.

Air, without liquid, in the pleural cavity is called pneumothorax ; with serum, it is termed pneumo-

hydrothorax; and with pus, it is known as pneumopyothorax.

On inspection, in pneumothorax, there is seen decided dyspnoea. But, inasmuch as the affected side is in a state of permanent expansion, with bulging of the intercostal spaces, from this influx of air, the respiratory movements are absent from that locality. Upon the unaffected side they are diminished. Consequently, the breathing is mainly diaphragmatic.

By palpation the vocal fremitus is found to be absent over the volume of air, and the heart moved by this gaseous effusion toward the normal side.

On mensuration, the half-circumference of the inflated side of the chest is commonly much increased, but it is not impossible for air to occupy the pleural cavity at the expense of the viscera alone.

By percussion, the resonance is tympanitic, whereof the pitch is sometimes high and other times low, according to the tension of the parietes produced by this elastic fluid; for, the greater the stretching, the higher the pitch.

On the other hand, wherever the lung chafes to be pressed, or is adherent, there will be dullness; and some thickening of the pleura may also cause dullness on light percussion; but with a forcible stroke,

under these circumstances, the tympanitic quality usually comes out.

By auscultation, the vesicular murmur is found wanting, but now and then bronchial breathing can

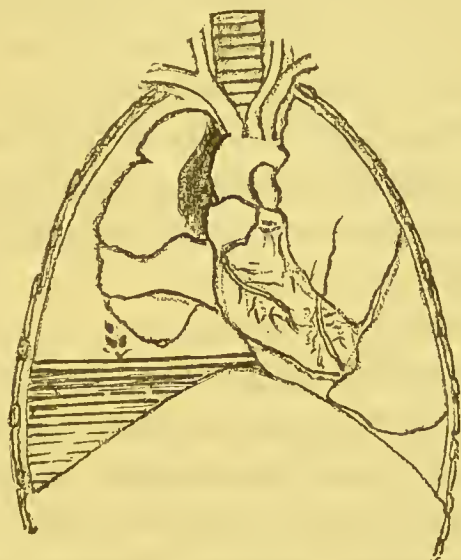


Diagram showing air and fluid in the right pleural cavity.

be heard, as from a distance, even where there is no fistulous opening; and when the channel through which the air entered the chest remains patent, there is well-defined amphoric respiration. Moreover, the metallic echo of the voice in the pleural cavity is of considerable significance; or, better, the metallic echo of a percussion stroke upon the pleximeter, which, however, requires the assistance of a third person.

Meantime, over the compressed lung there is bronchial breathing, and in the healthy lung there is puerile respiration.

Now, upon the advent of fluid in the pleural cavity, be it serous or purulent, the tympanitic resonance gives way to flatness, from below up as far as the liquid extends. Then there is tympanitic resonance above and flatness below the level of the effusion. And here, more particularly, a change in the patient's position causes a movement of the fluid that perceptibly alters the level of flatness.

Vocal fremitus remains absent. There is more or less fever associated with pneumo-pyothorax.

The auscultatory signs continue the same above, with the addition of an occasional metallic tinkle, while below the level of the effusion there are neither voice nor respiratory sounds. But the physical manifestations peculiar to this condition (air and fluid in the pleural sac) are splashing râles, developed by succussion, a method of investigation described in connection with auscultation.

SUMMARY : SIGNS OF AIR AND FLUID IN PLEURAL SAC.

Inspection : Immobility, expansion dyspnœa.

Palpation : Vocal fremitus absent.

Mensuration : Semi-circumference increased.



Calorimetation : Possibly fever.

Percussion : Tympanitic above, flat below.

Auscultation : Respiratory murmur absent ; or, amphoric breathing above, all sounds absent below ; splashing râles.

### PHTHISIS PULMONALIS.

**Acute Phthisis.**—So far as the physical signs are concerned, acute phthisis differs from chronic phthisis in the quickness with which these signs are developed.

Because of the rapid and extensive hepatization that is associated with the tubercular process, this malady has been termed pneumonic phthisis ; and, moreover, for the same reason, in the commencement of the disease, it is now and then mistaken for pneumonia, from a similarity in the signs and symptoms.

But in acute phthisis the frequency of the respirations is not so great as in pneumonia ; and, although the evening temperature may rise to 104° F. a number of times in the course of the affection, still it more often fails to reach that mark, and in the morning it is commonly not much above 100° F., while frequently it falls to 99° or 98·6° F. In addition, the crepitant râle is less prominent than a

subcrepitant râle. The dullness on percussion gradually changes, in the region of excavation, to amphoric or cracked-pot resonance, at the same time that bronchial breathing and bronchophony, heard in the vicinity of solidified lung, give way to gurgling râles, cavernous breathing, and pectoriloquy.

That the foregoing signs are, in some instances, confined to one side of the chest, is explained by the occasional post-mortem appearances, namely, miliary tubercles scattered throughout both lungs, with consolidation and vomicæ in but one lung.

**Chronic Phthisis.**—The first stage of phthisis is marked by the advance of deposition up to well-defined consolidation, and the second by a breaking down of the necrosed tissue with final excavation.

As this process begins at the upper part of the lung and travels downward, it is near the apex that the early signs of these changes become apparent.

In chronic miliary tuberculosis the little tubercles may be so scattered through the lung, or, if confined to the apex, so complicated by emphysema, as to nearly, if not quite elude detection.

#### *First Stage.*

*Inspection.*—There is probably very little about the contour of the chest to justify a suspicion of com-

mening phthisis. But early in the disease can be seen, upon careful inspection, some restriction of the inspiratory expansion at the apex of the affected side, and later there is more or less depression in this same locality. The lack of motion is more noticeable in women than in men, owing to the greater natural mobility in the superior costal region of the former. An increase in the frequency of the respiratory action is not especially pronounced, except upon exertion.

*Palpation.*—The diminished expansibility of the affected side is perhaps best appreciated in supplementing inspection by palpation, while the examiner stands behind and enough above the patient to look down upon his chest.

As a rule, the intensity of vocal fremitus increases with the consolidation; but, for reasons given elsewhere, this sign is less serviceable in women than in men; and it always has greater significance upon the left than the right side with both sexes, because of its habitual predominance in the latter situation.

The momentary circumscribed superficial elevation produced by a smart tap of the finger upon the chest, known as “myoidema,” is absent in so many cases of well-marked phthisis, and, contrariwise, present in such a number of patients where phthisis is

undemonstrable, that the writer is led not to consider it a distinctive sign of the malady, but rather a somewhat frequent coincidence. It seems to be oftenest found in the emaciated who have been, heretofore, decidedly muscular, and seldom in those suffering from wasting disease, be it phthisis or not, whose muscular development was never pronounced.

*Calormetation.*—Fever is one of the earliest physical manifestations of phthisis; and, although there is no thermometrical range peculiar to this malady, pyrexia is pretty sure to accompany the advance of the disease, and to disappear while it remains stationary. At some period, then, in the twenty-four hours fever can be found, and this is usually toward evening. Moreover, an intermittent moderate elevation of temperature indicates a slower progress of the lesion than a slight but constant pyrexia, while rapid strides may be inferred if the fever continues high; yet death is sometimes preceded by a subnormal temperature.

*Percussion.*—Dullness is found in degrees varying with the onward course of this stage, and is best developed above the clavicle by a gentle stroke while the patient's mouth is open. Upon the clavicle it is customary to perform immediate percussion, for here the bone takes the place of a pleximeter.

The surest way to render delicate variations of tone appreciable is to place the back of the patient against a sounding-board, and another available device is to practice respiratory percussion at the same time. It must be borne in mind, however, that the normal pitch is almost always higher at the right than at the left apex, and also that it is possible for a localized emphysema to hide the signs of tubercle.

*Auscultation.*—There are three different phases of the respiratory murmur that may obtain at the apex in early phthisis: 1. The expiration, either with or without the normal inspiration, may be prolonged and of high pitch. 2. The respiration may be extremely feeble in this locality. 3. The rhythm may be broken into little puffs, known as jerky or cog-wheel respiration, whether the pitch be high or low. But this jerky rhythm must not be confounded with a similar condition sometimes caused by the pulsation of the heart against the lung.

As a rule, the cardiac sounds become accentuated in the area of consolidation, and there may be a systolic murmur over the subclavian artery.

Now, when it is taken into account that vocal fremitus and vocal resonance are not always reliable signs, and further that the breathing is less pulmonary in quality at the right than the left apex, it

follows that the changes must be unquestionable on that side to be decisive. In fact, there should be some of the adventitious signs, of localized bronchitis, pleuritis, or parenchymatous inflammation, to insure a reasonably certain diagnosis.

Later in the disease either broncho-vesicular or bronchial breathing can be heard, and, if bronchial râles are present, they are usually high in pitch.

Finally, toward the end of this stage, depression, with lack of mobility in the affected region, grows well marked, but allowance must be made for a certain amount of excavation beneath the clavicle, due to a waste of tegumentary tissue; and, though vocal fremitus may reach a more decided intensity, it is at times obscured by an overlying pleuritic exudation.

The dullness now becomes unmistakable, and, when there happens to be much thickening of the pleura, this quality of resonance may be out of all proportion to the extent of deposit in the lung.

A rise in the pitch behind over the scapula is best detected by comparing the dullness of one bone with that of the other; for dullness is normal in this region, and only increased by an underlying consolidation.

Vocal resonance may be exaggerated, or bronchophony be heard; and, while the breathing by this time is commonly bronchial, it occasionally is inaudi-



ble, from the obstruction of a bronchus by collected matter, or on account of the general feebleness of the

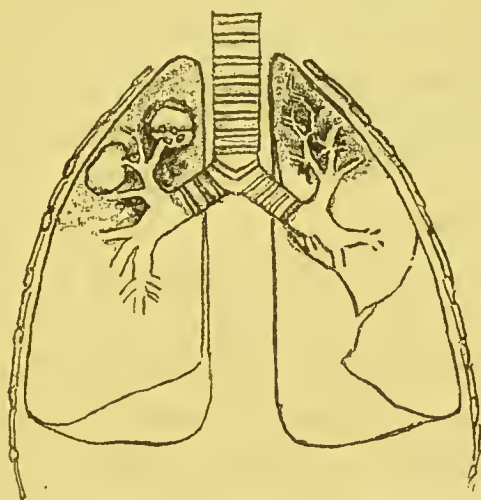


Diagram illustrating chronic phthisis in stage of consolidation on left and excavation on right side.

respiration. Under these circumstances a cough will at least develop a transitory bronchial puff in the one, or permanently restore the breathing in the other instance.

### *Second Stage.*

In the change from the first to the second stage there is softening of the inflammatory products, breaking down of the pulmonary tissue, and final evacuation of the necrosed substance.

Now, although there are no positive evidences

of softening until bronchial communication is established, yet, when sharp, crackling, moist râles are heard over the affected portion of the lung, small-sized vomicae probably exist, and therefore softening has taken place. Hence the physical signs of this stage are chiefly those of excavation. But a cavity must be fairly large before its presence can be conclusively shown.

*Inspection.*—The impairment of inspiratory expansion, and the depression below the clavicle, in many instances remain. But now and then mobility seems to be less restricted and depression more effaced than hitherto, possibly from the destruction of contracting tissue in a large superficial excavation.

*Palpation.*—Vocal fremitus at this stage, though variable, is usually increased.

*Percussion.*—The resonance depends upon the position of the cavity, its size, contents, and the nature of the intervening tissue. Over a small excavation there will be well-marked dullness, and the same holds when a large one is filled with fluid. Also, where considerable solidification of the lung or thickening of the pleura overlies a cavity, there will be dullness. A large empty cavity in close proximity to the surface of the chest may give tym-

panitic resonance; and, if the walls of the vomica are flexible, and there is a free channel from the cavity to the open mouth, a cracked-pot sound can be developed by strong percussion. Finally, light strokes often fail to bring out anything but dullness, where heavy strokes will elicit tympanitic, amphoric, or cracked-pot resonance.

*Auscultation.*—A medium-sized cavity may give only bronchial breathing, which later becomes cavernous, but which meantime is not infrequently a mixture of both kinds of respiration. If a collection of fluid obstructs the free entry of air, there will be gurgling râles. When the fluid is below the opening, cavernous breathing is audible, and gurgling râles if the patient coughs. On the other hand, vomicæ of considerable size, with tense walls and free openings into large bronchi, produce amphoric breathing. Metallic tinkling sounds are sometimes heard. Lastly, there is whispering pectoriloquy, or, what perhaps is more common, cavernous whisper—the difference being, if distinction is deemed necessary, that articulation in the latter is less defined than in the former.

In conclusion, it is important to know that the physical signs of a cavity, though absent in front, may yet be found in the axilla or the supra-spinatous fossa.

## SUMMARY: SIGNS OF CHRONIC PHTHISIS.

*First Stage.*

Inspection: Immobility with depression.

Palpation: Vocal fremitus increased.

Mensuration: Negative.

Calormetation: Fever.

Percussion: Dullness.

Auscultation: Long, high-pitched expiration; breathing feeble; cog-wheeled; adventitious râles; vocal resonance increased.

Later, bronchial breathing; bronchophony.

*Second Stage.*

Inspection: Immobility; depression same or less.

Palpation: Vocal fremitus increased.

Mensuration: Negative.

Calormetation: Fever.

Percussion: Dullness; amphoric; tympanitic; cracked-pot.

Auscultation: Cavernous breathing and whisper; gurgles; amphoric breathing, pectoriloquy, metallic echo.

**Acute Miliary Tuberculosis.**—A sudden rise in the temperature, decided acceleration of the pulse, and

marked frequency of the respirations, associated with more or less cyanosis of the lips and finger-nails, in a patient known to have phthisis, would strongly point to the supervention of acute miliary tuberculosis if upon physical exploration no other cause could be found for these manifestations.

The râles of a co-existing bronchitis are practically the only physical signs of discrete tubercle in the chest. But, as these little bodies are generally located in other organs as well, an ophthalmoscopic examination often reveals them in the choroid tunic of the eye.

Acute idiopathic miliary tuberculosis is not infrequently mistaken, and with reason, for typhoid fever, owing to their great likeness in appearance.

There is a somewhat more abrupt invasion of the first-named affection; it is not so often accompanied by diarrhoea, and the course of the fever is attended by irregularities—such, for instance, as pronounced morning, instead of the customary evening, elevations peculiar to typhoid fever.

**Fibroid Phthisis, or Interstitial Pneumonia.**—The distinguishing features of what is sometimes styled fibroid phthisis are those referable to the contraction of the lung, and, until such takes place, there is

little foundation for isolating this from other forms of phthisis by physical signs.

When, however, contraction does occur, there is a uniform falling-in of the affected side, associated with a relative expansion of the normal or less affected side. The heart is noticeably displaced in the direction of the retraction. There is dyspnœa upon the slightest exertion. Vocal fremitus may or may not be increased; and the half-circumference of the diseased side measures considerably less than that of the other. Fever makes its appearance toward evening. Dullness, with a marked feeling of resistance to the fingers on percussion, is wide-spread over the affected side, while over the area of expansion resonance is intensified, and almost vesiculo-tympanic in quality. Listening reveals puerile respiration in the last-mentioned region, but bronchial or at least broncho-vesicular breathing and bronchial râles are heard in the former, together with the various signs of vomicæ, when extrusion of necrosed tissue has happened.

#### PULMONARY GANGRENE.

A diagnosis of gangrene of the lung chiefly rests upon the peculiar fetid breath and sputum of the pa-



tient in whose lung consolidation and possibly excavation has taken place.

This sputum is of a dark-brown or dirty-gray color, very offensive, and sometimes contains shreds of lung-tissue. High fever and considerable dyspnœa may attend the disease.

It is to be distinguished from fetid bronchitis, and from phthisis with a passing fetor of the breath and sputa. Now, dullness is always absent from bronchitis, and, on the other hand, usually present in gangrene; and, although a temporary gangrenous odor may occur in phthisis, there will have been antecedent signs of vomicæ, whereas in sphacelus of the lung the bad smell is usually coincident with the first evidences of excavation.

#### CANCER OF THE LUNG.

It is impossible to demonstrate with any certainty by physical signs the existence of disseminated cancer of the lung. Where, however, there is a cancerous growth of considerable magnitude, the manifestations are less equivocal.

The new growth may start in the mediastinum, spread, but not equally, into both lungs, and in its course involve the pericardium; or, have its origin

upon one side of the chest, and there remain to the end.

Certain forms of cancer cause a contraction of the side most affected, but more or less bulging of the thorax is the rule, with impairment of inspiratory expansion. There is usually dyspnœa, as well as increased frequency of the respirations. Some displacement of the heart occurs, and there may be œdema and congestion of the head, neck, and arms. The blood-stained sputa have the appearance of red-currant jelly.

Vocal fremitus, though variable, is often wanting. In measurement the half-circumference of the affected side is sometimes much increased. Pyrexia is commonly absent.

Upon percussion, flatness extends with the tumor, and there is dullness beyond if the adjacent lung becomes inflamed. This flatness is attended by so much resistance to the fingers, that fluid might be suspected were it not for the locality; for an effusion almost invariably gravitates to the bottom of the pleural cavity, and anteriorly seldom exceeds the median line. Hence, if the resonance is pulmonary below and behind, while, on the other hand, the flatness reaches across the sternum in front, the latter is not due to a collection of liquid in the pleural cavity. But should

pleurisy complicate a mediastinal tumor, as it often does, the fluid must be drawn off before the graver malady can be diagnosticated and the growth outlined.

Upon listening in the area of flatness, the breathing is either bronchial or faintly vesicular, or else entirely suppressed. The signs caused by compression of a bronchial tube vary with the amount of the obstruction from sonorous breathing to complete silence. Where the lung breaks down and extrusion follows, there are evidences peculiar to a cavity.

Finally, though an intra-thoracic tumor in the course of the aorta may simulate an aneurism, still the transmitted pulsation lacks the expansile quality found in the latter; and, moreover, the history and symptoms of the two affections help to distinguish one from the other.

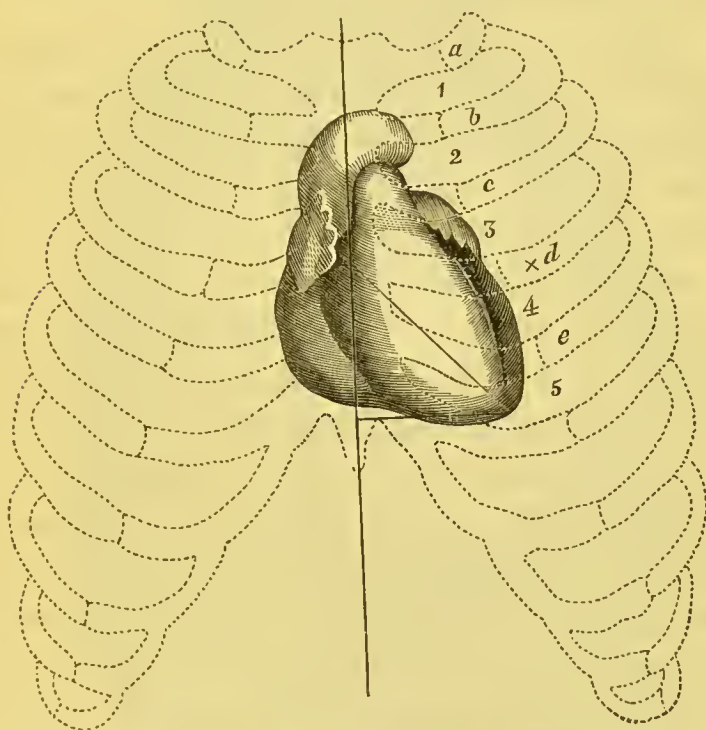
## EXPLORATION OF THE HEART.

THE relative position of the heart to the surface of the chest is differently stated, according to the circumstances under which observations are made—whether, for instance, the specimen is frozen or not at the time the measurements are taken.

But, for practical purposes, it is sufficiently accurate to consider that the organ, mostly covered by lung, lies obliquely in the chest, with its base upward and backward to the right, and its apex downward and forward to the left. The upper border rises a little above the level of the sternal insertions of the third costal cartilages; the right border extends about an inch to the right of the sternum; the left border nearly reaches the left nipple line; and the lowest point of the heart approximates the upper edge of the sixth left costal cartilage.

While the superficial cardiac region, or that uncovered by lung, is variously described by different writers, it is in fact irregular in shape, of variable size

in diverse persons, and on the whole not of much real consequence, for percussion of the heart must



Heart *in situ* (Dalton, in Flint, "on the Heart"). *a*, *b*, *c*. etc., ribs; 1, 2, 3, etc., intercostal spaces; vertical line, median line; triangle, superficial cardiac region;  $\times$  on the fourth rib, nipple.

include not only this part, which gives flatness, but also that beneath the lung, which gives dullness, to be of definite utility.

Although an enlargement of the heart may increase this area of flatness, a retraction of the left

lung will do the same ; and it is temporarily made smaller and larger, alternately, by inspiration and expiration respectively.

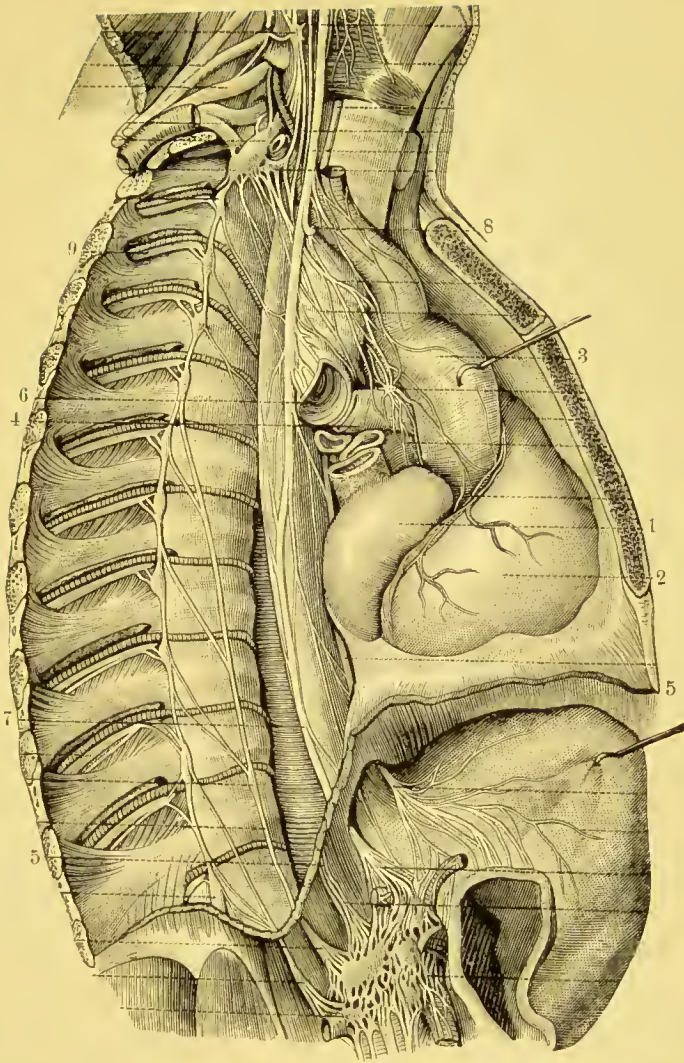
This region of flatness, however, may extend to the limits of the pericardial sac, in which there is an effusion ; and, conversely, an emphysematous lung, when distended to a great degree, may quite obliterate the superficial space.

**Arteries.**—The arch of the aorta passes obliquely upward and forward behind and toward the right edge of the sternum, thence to the left and backward along the left side of the third and down the left side of the fourth dorsal vertebra. At this point the vessel becomes the thoracic portion of the descending aorta, which extends along the left side of the spine, then in front, and terminates at the aortic opening of the diaphragm. The transverse part of the arch rises to within an inch of the suprasternal notch, which in some instances it nearly reaches. The pulmonary artery is located behind the sternum, from origin to bifurcation, beneath the arch of the aorta.

The *arteria innominata* ascends slanting from the arch of the aorta to the upper margin of the right sterno-clavicular articulation.

**Relative Site of the Valves.**—The pulmonary valves are situated behind the junction of the third left cos-





THORACIC CAVITY. VIEW OF HEART AND BLOOD-VESSELS  
FROM THE RIGHT SIDE (SAPPEY).

- 1, right auricle; 2, right ventricle; 3, arch of aorta; 4, pulmonary artery; 5, section of the diaphragm; 6, section of right bronchus; 7, right pneumogastric nerve; 8, arteria innominata; 9, intercostal vessels and nerves.



tal cartilage with the sternum ; the aortic, underneath the third intercostal space, a little below the pulmonary, and nearer the median line ; the mitral valve, deeply beneath the third intercostal space, and nearly an inch to the left of the sternum ; the tricuspid, behind the sternum, on a level with the fourth costal cartilage, and about in the median line.

**Heart-Sounds, and where heard.**—It is important that heart-sounds should be disassociated in the student's mind from heart-murmurs. Sounds are normal and therefore audible in health, whereas murmurs are abnormal, and hence occur only in disease. The sounds, to be sure, may be heard both in health and disease, but under the latter circumstances they are modified in quality.

Owing to the close proximity of the valves of the heart to one another, these sounds are best distinguished at points somewhat removed from their origin. Such positions are found in the direction of the blood-current, and also where the heart approaches the surface of the chest.

The aortic sounds are carried by the onward flow of blood to the second right intercostal space, close to the sternum ; the pulmonary to the left edge of this bone, in the same relative position. On the other hand, the sounds of the mitral valve are conducted by

the left ventricle to where the apex comes in contact with the thorax ; while those of the tricuspid are con-

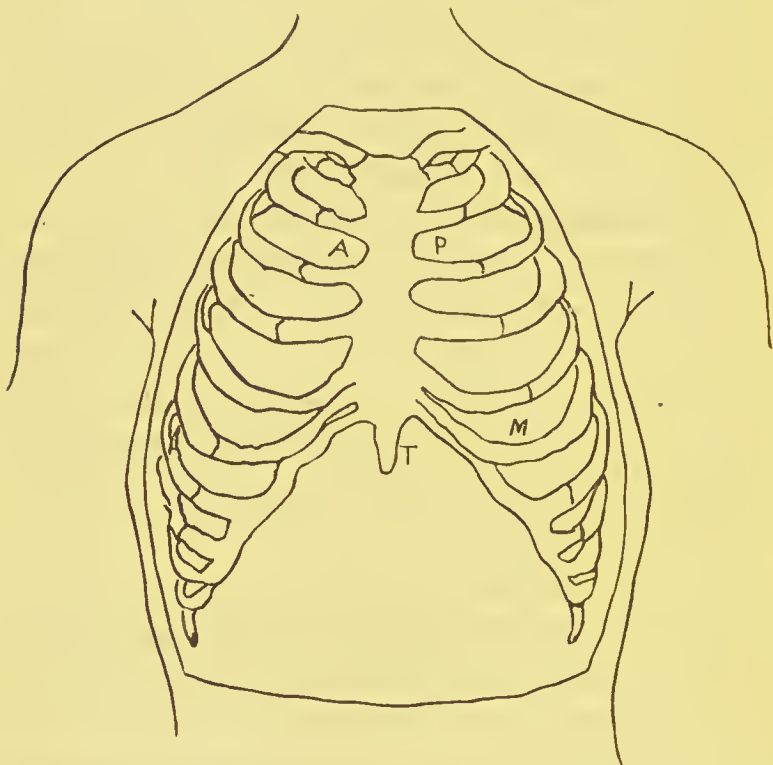


Diagram showing the positions for listening to the heart-sounds. A, aortic ; P, pulmonary ; T, tricuspid ; M, mitral.

veyed directly through the right ventricle, at the lower part of the sternum. Here are the points of maximum intensity, not only of heart-sounds but also, as a rule, of valvular murmurs.

Now, what is the accepted explanation of the mechanism of heart-sounds? The first sound, which is synchronous with the apical impulse, is produced by the closure of the auriculo-ventricular valves, by contraction of the muscular fibers of the heart, and by the impulse of the organ against the chest. The second sound is due solely to the sudden closure of the aortic and pulmonary valves. First, a sound, then a short silence, followed by a second sound, and then a longer silence, comprise rhythmic cardiac action. Both these sounds are audible at the apex, and both are heard also at the base; but, in the former instance, the accent is upon the first, and in the latter upon the second sound.

In order to fully appreciate this cycle of the heart's action, let the student for a moment consider what is taking place within the organ. While blood is being thrown from the right ventricle into the pulmonary artery, and from the left ventricle into the aorta, the auricles are slowly filling. Then, upon closure of the aortic and pulmonary valves, blood flows passively into the ventricles; and, just before the first sound of the heart, auricular systole drives the remaining blood from the auricles into the ventricles, which in turn propel it onward, one into the pulmonary and the other into the systemic circulation.

The systolic contraction or wave, if it may be so termed, really begins in the great veins at the en-

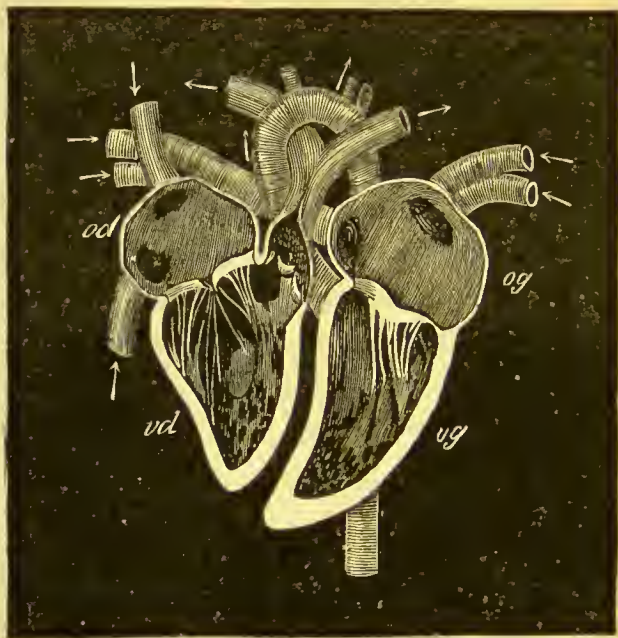


Diagram of the four cavities of the heart (Bernard). *od*, right auricle; *vd*, right ventricle; *og*, left auricle; *vg*, left ventricle. The arrows indicate the course of the blood.

trance to the heart, spreads through the organ, and passes on into the large arteries.

**Methods of examining the Heart.**—The same methods serve to explore the heart that are employed in examination of the lungs.

By inspection the point where the apex of the



heart strikes the chest is shown, and also, to some extent, the nature of the impact. The normal site of this pulsation is commonly two inches from the sternum in the fifth left intercostal space; still, it is safe to extend these limits to the sixth left rib below, and as far as the mammillary line. In some instances, from enlargement of an auricle or retraction of the lung, or both combined, there is visible cardiac pulsation in the third left intercostal space. And an increase in the size of the right ventricle will produce a beating in the epigastrium. But neither pulsation in this region, nor its absence over the apex, is of necessity abnormal.

There are two or three things that alter the position of the apex which are not included in the category of disease—namely, posture, distention of the abdomen, and respiration. First, the apex falls toward the side upon which the body rests; second, it is elevated by a full stomach, or by the pressure due to pregnancy; third, it is depressed by a deep breath—all of which must be taken into account at the time of examination. But, aside from these, considerable displacement may be caused by disease elsewhere than in the heart. For instance, an intrapleural effusion will push the apex from the affected side, while, on the contrary, it is dragged toward the side wherein

there is a retraction of the lung. An enlargement of the left lobe of the liver may crowd the apex to the left. It is pressed downward to the right, and sometimes hidden by an extensive pulmonary emphysema. There is an apparent but not real elevation of the apex in the early part of a pericardial effusion, owing to a transference of pulsation to another part of the heart. These likewise must be excluded before other interpretation is given to a removal of the apex from its wonted place.

Finally, it is carried to the left and downward by enlargement of the left ventricle; to the left, and possibly away from the chest-wall, by a similar condition of the right ventricle; and downward, as well as to the left, by an increase in size of the whole organ. Moreover, the displacement is in direct proportion to the enlargement, and the quality of the impulse to the nature of the increase.

In simple hypertrophy of the left ventricle, the apex strikes the chest with more than normal force; in hypertrophy of the right ventricle, the apex exceeds the nipple line; but here the force of impulse is transferred to the epigastrium, and comes from the affected ventricle. When the walls of both ventricles are thickened, though the pulsation is strong, and there is perceptible heaving of the entire præ-

cordia, the shape of the heart is altered, and the stroke of the apex is not well defined. But in dilatation the pulsation is weak, diffused, and undulating, or else quite invisible. Furthermore, there are many grades of dilated hypertrophy between these two extremes of hypertrophy and dilatation, in which the impact is either weak or strong, as one or the other predominates.

Still, these changes in the position and force of the apical impulse are determined with far greater accuracy by the hand than the eye, and by palpation, too, valvular thrill and pericardial friction can be appreciated.

The actual distance between points established by other signs is found on mensuration.

Thermometry helps the examiner to decide between acute and chronic heart affections, for the latter are usually unaccompanied by fever.

*Percussion.*—Inasmuch as enlargement of the heart increases the area of the præcordial flatness and dullness in the direction of the enlargement, percussion will define the extent of this increase. Therefore, it is important to know where percussion can be performed with advantage, and also what are the limits of normal cardiac resonance.

The two positions best suited to this purpose lie

respectively one inch to the left of the sternum, in the parasternal line, and across the chest, upon a level with the fourth rib. Commencing, then, to percuss in the parasternal line from above downward, there is a rise in pitch that amounts to dullness at the lower border of the third rib, which changes to flatness at the lower edge of the fourth, and thus continues into the left hepatic area. Next, from without inward, the resonance becomes dull just within the nipple line, and so continues to the free border of the lung, and thence over the superficial area of the heart there is flatness as far as the sternum. With an increase of the right side of the heart, dullness can be made out at the right of the sternum by percussing from without toward this bone at that same level. But in health the results of percussion in this region, as well as over the sternum, are not altogether satisfactory. Still, having located the apex and defined the upper and left borders of the heart, the size of the organ can be quite accurately estimated.

The region of cardiac flatness does not exceed the fourth costal cartilage above, nor the sixth below, the parasternal line on the left, nor the left edge of the sternum on the right.

A decrease in the size of the heart is not readily

made out, but pulmonary emphysema may so lessen the extent of the dullness and flatness of that viscus as to convey the impression that atrophy has occurred. And, what is more, great distention of the lung will mask the signs of an existing enlargement of the heart.

*Auscultation.*—The sounds and likewise the murmurs of the heart are revealed by auscultation.

If a stethoscope is placed over the apex, which is the mitral area, a dull, prolonged tone is heard, low in pitch, and accentuated; this is followed by a brief pause, and then by a short, sharp, high-pitched note, that in turn is succeeded by a somewhat extended silence. This is the circuit of one cardiac revolution, with systole and diastole as heard in the mitral region. Upon the adjustment of the instrument to the aortic area, close to the sternum in the second right intercostal space, these two sounds are again heard, and with rhythm unchanged, but with the accent transferred from the first to the second sound.

Now, though both valves at the base unite in the formation of the second sound, and each of the auriculo-ventricular valves contributes its share to the first, yet these sounds are separable one from another. There is more intensity to the click of the aortic than the pulmonary valves, notwithstanding

the latter are nearer the surface of the chest than the former. And, while the sound of the tricuspid valve is higher in pitch than that of the mitral, it has decidedly less accentuation. Moreover, there is, in many instances, a neutral point between these two areas, where the sounds vanish, or at least become less distinct, and beyond which they reappear, to be again plainly audible.

Seeing that the first sound of the heart is synchronous with the impact of the apex against the chest, when there happens to be no perceptible shock, this sound is determinable, should there be a difficulty in its recognition, by placing the fingers upon the carotid artery, which also pulsates in synchronism with cardiac systole. And the position of the apex can be found, under these circumstances, with the stethoscope, by noting the seat of greatest intensity in the first sound.

Now and then in women with large *mammæ*, or in very fat persons, it is not easy to map out the superficial cardiac space by percussion, but through auscultation the borders of the region can be traced with facility, by moving the stethoscope along the lung in this vicinity while the patient speaks; for vocal resonance will come to an abrupt termination at the margin of the lung, which marks the limitation of the sought-for place.



**Modifications of Sounds.**—There is a regular intermission of the first sound of the heart in some persons which is not necessarily incompatible with health. In others there is a reduplication of the second sound, from a lack of simultaneous action of the two sides of the organ, which points to an obstruction in the circulation, and commonly at the mitral valve.

The first sound is dull and muffled at the apex in simple hypertrophy, and is characterized more by a throb than a sound; but with dilated hypertrophy it is clear, loud, and far reaching; while in dilatation it is sharp, high pitched, and at the same time very weak. Indeed, it is feeble, not only in cardiac dilatation, but also with fatty degeneration of this organ, and, in fact, with all diseases that occasion failure in the contractility of the ventricle. And, moreover, a feebleness of the valvular sound at the left apex, in comparison with that of the right side, may indicate a mitral affection. Yet it is well to bear in mind pulmonary emphysema as a possible cause of this, for the distended lung may both stifle the sound of the mitral valve, and render the tricuspid more intense, through a consequent hypertrophy of the right ventricle upon the one, and the covering up of the left upon the other hand.

The second sound at the base is generally intensi-

fied by hypertrophy and weakened by dilatation. It is deprived of strength by valvular lesions that diminish the quantity of blood in the arterial system, such as mitral stenosis or insufficiency; and, at the same time, as well as from a like cause, the pulmonary second sound becomes accentuated through augmented pressure in the venous system. Hence, though there is always a possibility that the aortic second sound, enfeebled by disease, may render the pulmonary apparently but not really stronger, yet, with this prevision, when there is no disease of the lung to account for a persistent accentuation of the pulmonary second sound, such a condition may be taken as presumptive evidence of a valvular affection, and more especially of the mitral orifice.

**Heart-Murmurs.**—A heart-murmur is an adventitious vibration set up within this organ, and sometimes extending into its immediate outlets, by the production of a sonorous rush in the blood-current.

This is brought about by an abnormal narrowing of the canal, through some obstruction, which accelerates the velocity of the stream, and throws the fluid into eddies beyond, where the clashing particles become audible; and the loudness of this murmur depends in part upon the force with which the current is propelled.

Let the student take a soft-rubber tube, say two feet long and one inch in diameter, through which water is being driven with considerable power, and he will find an example of this sonorous wave, as well as of the variation of intensity with the propulsive force; for, wherever he interrupts the stream by squeezing the pipe, he will hear a murmur, with his ear upon the tube, and this murmur will be most perceptible at the further side of the compression. Moreover, it will be loud or soft as the pressure of the current is raised or lowered.

Now this same thing happens when changes occur in the valves that agitate the otherwise noiseless flow of blood. For instance, a contraction of the mitral orifice scatters the stream in the left ventricle; and a like condition at the aortic aperture dissipates the current in the ascending portion of the aorta. So, too, a reflux at the mitral opening is thrown into audible vibration in the left auricle by the imperfectly closed valve, which is practically an obstruction; and in a similar manner regurgitation from the aorta is attended by a sonorous jet into the left ventricle.

Just how an anæmic bruit is produced, however, is not so clear; but it is assumed that the aorta and pulmonary artery at their commencement do not contract in proportion to the reduced volume of the

blood, and that tumultuous vibrations are developed from the passage of the fluid into these relatively dilated cavities.

Although lesions of the valves that cause stenosis, patency, or both at once, usually give rise to murmurs, it does not follow that all murmurs of the heart have this significance, nor that some of these apertures may not be seriously affected without a manifestation of this kind. Nevertheless, the coincidence of a bruit and a lesion is sufficiently common to warrant the establishment of such a rule. But in view of the fact that it is far easier to detect these noises than to determine their purport, the student should never fail to scrutinize the whole heart in his endeavor to solve the meaning of a murmur. Wherefore, it is essential to know what effect a valvular disease may have upon the cavities of this organ, for certain modifications are sure to follow (in a degree conservative), which help to substantiate the diagnosis. Thus, with stenosis of the aortic orifice, there is hypertrophy of the left ventricle; with a like contraction of the mitral aperture, there is a dilated hypertrophy of the left auricle, and also in the end of the right side of the heart; with insufficiency of the aortic valves, there is dilatation as well as hypertrophy of the left ventricle; with the same failure in the competency of

the mitral valve, there are dilatation and hypertrophy, first of the left auricle and then of each ventricle ; and with both stenosis and insufficiency at the aortic opening, the entire heart eventually becomes excessively enlarged.

Still, in spite of all that, so long as the hypertrophy keeps pace with the extra requirements of the heart, the disease is somewhat in abeyance, and the lesions are said to be compensated ; but, when hypertrophy lags behind dilatation, there comes a rupture in the established equation — a danger-signal which betokens the approaching fatal termination of the malady.

The quality of a murmur can not be said to be an index to the gravity of a lesion, since some bruits are loud, others soft ; one harsh, and another cooing ; yet at the same time a certain amount of information can be gained thereby.

It is evident, from the manner in which a bruit is developed, that, if the muscular tone of the heart is good, there will be more intensity to the sound than when the organ is weak. It is also plain that, where regurgitation is extensive, the duration of the murmur will be shorter than where the return current meets with considerable obstruction in a valve not wholly incompetent. Then, too, where a portion of the valve

does its work, a modicum of the sound is often audible through the murmur. Furthermore, the examiner receives no little aid in diagnosis from the fact that the bruit of mitral stenosis is invariably rough and grating, that of anæmia always soft and blowing; also, that where two cardiac murmurs coexist, with a wide diffusion, there is frequently enough unlikeness to establish their separate origin.

Notwithstanding these few points, however, with a given murmur, the question presented is not so much in regard to its quality as to the integrity of the heart-muscles. Is there sufficient hypertrophy to compensate for the lesion, or has secondary dilatation occurred, and the disease begun its downward course?

There are three things to be taken into account with respect to a murmur subsequent to its discovery, namely, the point of maximum intensity, the rhythm, and the area of diffusion; but attention will be given to these further on.

A valvular bruit takes the place or part of the place of a valvular sound, or else precedes it. And in the genesis of a murmur there is a silent stage, when the valvular sounds are more or less muffled; then a transition stage, when the sounds might be called *murmurish*; and, finally, a stage wherein the bruit is unmistakable.

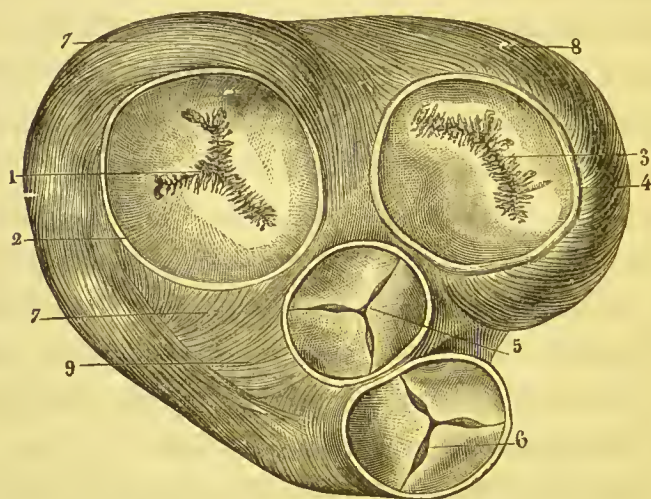


Sometimes a murmur that is inaudible while the patient stands is clearly audible when he lies; and, contrariwise, a murmur that can not be heard while the patient lies may be easily heard when he stands. Exercise not infrequently rouses an otherwise dormant murmur. Medication often restores the muscular power of the heart, and thereby generates a bruit where there was silent leaking. Lastly, in the impact of the heart against the lung, the inspiratory murmur may be so divided into little whiffs as to resemble an endocardial bruit; but such a source of error can be eliminated by causing the patient to hold his breath for a moment during the exploration.

**Systolic Murmurs.**—Before turning to the physical manifestations of heart-disease in their entirety, it will be of service to consider the bruits by themselves, and also their probable field of action. Reference has already been made touching the discovery of a murmur; and, inasmuch as disease is more prone to settle upon the valves of the left than upon those of the right side of the heart, we have chiefly to deal with bruits having an origin in that part of the organ.

There are two murmurs possible for each orifice, or eight in all, of which four, namely, mitral systolic, mitral presystolic, aortic systolic, and aortic diastolic, are most likely to occur, and with a frequency about

in the order of their enumeration ; and, the necessary changes being made, a like distribution applies to the



Valves of the heart (Bonamy and Beau). 1, Right auriculo-ventricular orifice, closed by the tricuspid valve ; 2, fibrinous ring ; 3, left auriculo-ventricular orifice, closed by the mitral valve ; 4, fibrinous ring ; 5, aortic orifice and valves ; 6, pulmonic orifice and valves ; 7, 8, 9, muscular fibers.

right side, yet a pulmonary lesion is almost unknown, except as a congenital affection, while disease of the tricuspid valve is only less rare.

Every murmur is determined by the time of its occurrence, the direction which it takes, and the location of its greatest intensity. Now the blood is driven from the left ventricle, during systole, through the aortic orifice ; and, meanwhile, all communication with

the auricle of this side is cut off by a closure of the mitral valve. But should the current encounter an obstacle at the aortic opening to its onward course, it would be thrown into confusion in the aorta, from which a murmur would arise and be carried upward. Hence this bruit is loudest at the aortic area, systolic in rhythm, and extends in the direction of the carotids.

On the other hand, should the mitral valve fail to close at this time, the blood would escape into the left auricle, as well as run through the proper channel, and be set in vibration by the impeding flaps at the mitral orifice. Here the bruit generated by this disturbance is borne with the reflux into the auricle, and thence to the back, and also by conduction through the apex to the front. Moreover, it is loudest in front and at the apex, because the heart is nearer the anterior than the posterior surface of the chest. Therefore this murmur is most intense at the mitral area, systolic in rhythm, commonly diffused to the left, and often audible near the inferior angle of the left scapula.

In a similar manner, during systole, the blood is being propelled by the right ventricle through the pulmonary aperture, and, likewise, the tricuspid valve is closed or very nearly so. Thus, supposing that an

obstruction were to occur at the pulmonary orifice, there would be a systolic murmur, with point of maximum intensity in the pulmonary area, and extension upward to the left, but not into the carotids. Still, practically, this need not occupy our attention.

So, too, in the event of tricuspid insufficiency, part of the blood would flow back into the right auricle, and give rise to a systolic bruit, best heard in the tricuspid area, and spreading upward to the right, yet not far.

An anæmic murmur is always systolic in rhythm, loudest at the base of the heart, and often as audible in the aortic as the pulmonary area. Furthermore, with anæmia pure and simple there should be no cardiac hypertrophy.

Now and then a systolic bruit is evolved from a disorder in the dynamics of the heart. The writer has seen an example of this, wherein the papillary muscles seemed to be, as it were, thrown out of gear, and the valvular flaps made inadequate by an epileptiform seizure. A young man was overcome by a spasm while being examined with a stethoscope, in whom a systolic murmur appeared over the left ventricle that was not present before the attack, and that disappeared in the subsidence of the spasm.

Not infrequently a systolic sound, closely simu-

lating a murmur, is audible in the tricuspid area, especially when there is a depression at the lower end of the sternum. This is attributed by Walshe to friction of a little white patch often found post mortem on the surface of the right ventricle.

Upon several occasions, more particularly when some obstruction already existed in the pulmonary circulation, from emphysema, for instance, the writer has heard a temporary tricuspid systolic bruit, which was brought about by the superadded pressure of a held inspiration, and was undoubtedly due to a transient dilatation of the right auriculo-ventricular orifice.

Lastly, when from any cause the cardiac chambers become dilated, and the valves are thereby rendered insufficient, systolic murmurs may appear, which, however, will vanish if mural integrity can be restored.

**Diastolic Murmurs.**—In diastole the aortic and pulmonary valves are closed, and the auriculo-ventricular valves open, while blood is flowing from the auricles to the ventricles. The vermicular contraction, styled cardiac systole, which was initiated in the veins and taken up by the auricles, has gone through the ventricles and reached the large arteries, wherein the recoil of the current finds a point of support at the closed semilunar cusps.



But, if the function of one or more of these cusps in the aortic valve be destroyed, each contraction of the artery will drive a portion of its contents back into the left ventricle; and the vibrations generated in this return-stream against the disorganized valve will cause a bruit that is aortic in origin and *diastolic* in rhythm.

Now, though this murmur of insufficiency is conveyed along the arteries a varying distance in the efflux, its main direction is backward with the reflux; still, not so much in the line of the ventricle as down the sternum, owing to the close proximity of this bone to the aortic valves, and its superiority over the heart as a conducting medium of sound. Furthermore, the point of maximum intensity of this bruit is oftener at the lower end of the sternum than in the second intercostal space.

Upon the other side, granting that the same thing could happen to the pulmonary valves, a diastolic murmur would be audible in the pulmonary area, but with an extension downward only.

**Exceptions.**—Although an aortic systolic murmur is loudest in the second right intercostal space close to the sternum, and a diastolic bruit at the lower extremity of this bone, yet, in some instances, these murmurs are heard only at mid-sternum, about on a



level with the third costal cartilages, and in others they are most intense in the second, and even the third intercostal space, close to the left edge of the sternum. In consequence of this, upon the exclusion of aneurism, a bruit within these precincts is presumably aortic and not pulmonary, especially if the right ventricle is unenlarged.

**Presystolic or Auricular Systolic Murmurs.**—The relatively passive flow of blood from auricle to ventricle, during the second cardiac silence, receives a sudden impetus, toward the close of this silence, by the contraction of the auricle; and should there be an abnormal narrowing of the mitral orifice, the impediment to the current, in addition to its increase of velocity, would throw the stream into sonorous jets.

Thus a murmur becomes audible a little above the apex of the heart, and just before the systole of the ventricle, that is termed mitral presystolic, but which in reality is auricular systolic in rhythm. This bruit is conveyed neither to the back nor to the left, nor yet very far in any direction.

A like affection of the tricuspid aperture would result in a murmur of the same rhythm, and be confined to that area; but, clinically, such a disease is rarely met with.

Now and then, as first explained by Flint, an auricular systolic murmur is audible in the mitral area that is due, not to stenosis of this orifice, but rather to the vibrations set up by the valvular flaps, which have been floated into contact by the blood returned from an aortic regurgitation, and which are suddenly driven apart by the auriculo-ventricular current. The writer has seen two or three cases that apparently were of this description.

**Pericardial Murmurs.**—The bruits of the pericardium, more properly termed friction, are developed between the visceral and parietal layers of this membranous sac, during either systole, diastole, or else both movements of the heart, but with the sounds of which they are not continuously synchronous. This friction, from a roughening of the pericardium, is often double, always superficial, and frequently transient. Moreover, though the rubbing is seldom heard over a wide area, it extends at times quite far, but equally in all directions, rather than chiefly, as with many endocardial murmurs, in the course of the blood-stream; and it is loudest when the body is bent forward, whereas valvular bruits are not particularly influenced by this posture.

A pericardial is distinguished from a pleuritic

friction mainly by the time and locality of its occurrence. Grating in the pericardium obviously is limited to the præcordial region, and is regulated by the action of the heart. That of the pleura is most prone to take place in the infra-axillary regions, where pulmonary mobility is extensive; and, further, it is dependent upon the respiratory movements.

**Associated Murmurs.**—The valvular bruits may be variously combined: thus, an aortic systolic with an aortic diastolic; mitral presystolic with mitral systolic; mitral presystolic with tricuspid systolic; aortic with mitral systolic; and, also, two murmurs at the aortic may be united with two at the mitral area.

**Venous Murmurs.**—The bruits produced in the veins of practical importance are those accompanying anæmia. Such murmurs are possible without this affection, but the affection seldom occurs without these murmurs.

In quality they are blowing, cooing, and sometimes musical; and, from the not infrequent resemblance of the noise to that of a humming-top, it has been denominated *venous hum*.

While heard elsewhere in the veins, the sound is usually most distinct at the lower third of the external jugular veins, and in the right than the

left side. It is always continuous in rhythm, but the intensity is often remittent, because of the periodical acceleration of the stream by the action of the heart. The direction is downward and inward along the subclavian and right innominate veins, so that it is now and then audible through the aortic area, and can be separated, with a little care, from the aortic sounds as well as from the respiratory murmur.

Where there is a question as to whether or not a given bruit is venous or arterial, pressure upon the vein above the stethoscope will stop the downward current and silence the venous hum; but, unless more force is employed than is required for this purpose, it will have no effect upon a murmur in the artery.

Now, the valves of the veins are encircled by little fibrous rings, which cause more or less contraction, at these points, in the caliber of the vessels. May it not be an unusual narrowing of this kind in the channel that throws the venous blood into vibrations beyond, and thus accounts for the bruits which are said to be heard where anæmia does not exist?

There is a valve in the external jugular vein an inch and a half above the inner end of the clavicle;

moreover, in this vicinity, the vein passes between the layers of an aponeurosis, to which it is attached, and by which it is held apart. Thus the vein, always less elastic than an artery, is unable to accommodate its capacity, owing to the adhesions, to the diminished bulk of the anæmic blood; hence the hypothesis that the stream, meeting with an obstruction at the orifice of the valve, is thrown into sonorous vibrations in the relatively dilated cavity at the further side; and, inasmuch as the venous current is increased in its rapidity by its near approach to the heart, as well as by the force of gravity, and also, to some extent, by the suction power exerted through inspiration, this is just where the bruit should be loudest.

A similar disturbance can be brought about, in some instances, by gentle pressure with the stethoscope upon the vein. So too, an enlargement of the thyroid gland may have this same effect.

## DIAGNOSIS BY PHYSICAL SIGNS OF DISEASES OF THE HEART AND OF THORACIC ANEURISM.

### ENDOCARDITIS.

AN acute inflammation of the endocardium is more prone to affect the left than the right side of the heart, and to settle upon the mitral than the aortic valve. It occurs most frequently with rheumatic fever, and reveals itself by a soft, blowing murmur that, as a rule, is located in the mitral area, with an extension to the left, and which is systolic in rhythm.

Still, should a systolic bruit arise at the base of the heart during the progress of this disease, though possibly hæmic, it might come from an affection of the aortic valve; and, furthermore, were a diastolic bruit to appear, it would be conclusive evidence that one or more cusps had been destroyed.

Whenever there is a sudden rise of the temperature in the course of rheumatism, the examiner must



not fail to include the heart in his search for the source of this elevation.

The possibility of an old valvular trouble with a new rheumatic seizure should be entertained, yet it usually can be dismissed if there prove to be no cardiac hypertrophy.

On the other hand, the supervention of an acute upon a chronic valvulitis is rather difficult to determine by physical exploration.

Acute ulcerative endocarditis happens in connection with blood-poisoning, and is liable to be masked by inflammatory affections of the pericardium, lungs, or pleura, due to the same cause. Hence the appearance of a systolic or a diastolic blowing murmur in a septic disease of low type would warrant the diagnosis of an ulcerative form of valvulitis.

It is claimed by Balfour that bruits heard during acute rheumatism are often the result of dilatation of the cavities from a relaxation in the heart-muscles, with a consequent failure in the adjustment of the valvular flaps, and that such bruits disappear when convalescence is established.

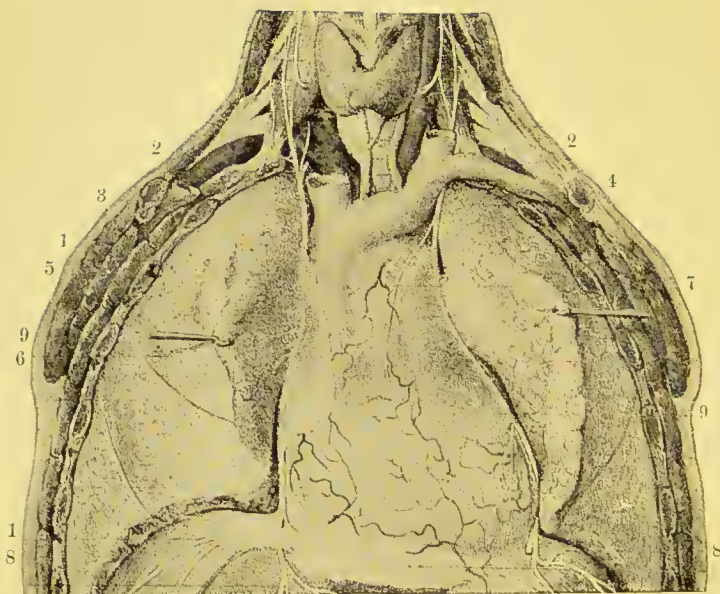
But this malady is not the sole cause of valvulitis, for the inflammation may be chronic from the commencement, obscure in its origin, and eventually lead to results quite as disastrous; and then, too, a founda-

tion may be laid in acute rheumatism that at first only modifies the valvular sounds, but which later reaches unmistakable proportions.

The chief interest in endocarditis centers upon the bearing it may have upon the future history of the heart. Now, to non-progressive and to slowly advancing lesions, this organ accommodates itself just as the body does to moderate impairments of function elsewhere, and the patient's existence is only slightly if at all imperiled or even incommoded; whereas, with rapidly advancing lesions, on the contrary, there very soon comes a disturbance of the balance that nature endeavors to maintain, wherein compensatory hypertrophy gives way both to dilatation and to mural decay.

It will be seen, therefore, in dealing with chronic heart-disease, that it is of considerable consequence for the examiner to recognize the difference between static and progressive lesions; and many times this may be accomplished by a careful inquiry into the history and symptoms, and by a scrutiny, withal, of the cardiac cavities.





THORACIC CAVITY.—LUNGS DRAWN APART. FRONT VIEW  
(HIRSCHFELD).

- 1, 1, pericardium ; 2, 2, subclavian arteries ; 3, trunk of the right innominate vein ; 4, trunk of the left innominate vein ; 5, superior vena cava ; 6, right phrenic nerve ; 7, left phrenic nerve ; 8, 8, diaphragm ; 9, 9, portions of the mediastinal pleura.

## PERICARDITIS.

An inflammatory affection of the pericardium is rarely idiopathic, but it is freely disposed to complicate rheumatism, pleurisy, disease of the kidneys, and kindred diseases. Moreover, it is often not apparent save by physical examination, and this is especially the case in chronic nephritis. Of these maladies, pericarditis is most commonly associated with rheumatic fever.

The first positive evidence that the pericardium is inflamed is gained by auscultation. A *friction* is heard over the heart, that may be a single rub or a to-and-fro grazing sound, which conveys the impression of being quite superficial. This sound may be loud or soft, and circumscribed or far-reaching; but it differs from most endocardial bruits in being diffused equally in all directions, rather than mainly in the course of the blood-stream. Furthermore, though it occurs oftener between the heart-sounds than with them, yet it may be so loud as to practically obscure them.

Unlike valvular disease, pericarditis has no effect upon the pulmonary second sound. And, finally, friction can be intensified by pressing the stethoscope against the præcordial region, by causing the patient

to lean forward, and by his taking a deep breath; whereas, these expedients have little if any influence upon endocardial murmurs.

Fever is present, but seldom ranges beyond  $102^{\circ}$  F.

If, by chance, rubbing should take place between the pericardium and an inflamed pleura, and continue

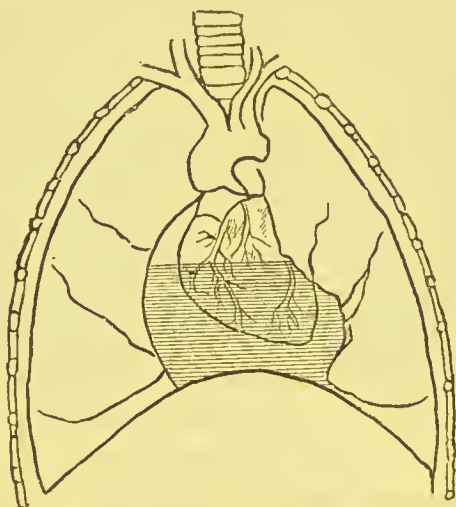


Diagram showing fluid in the pericardial sac.

even when breathing for a moment is stopped, as it now and then will do, the diagnosis of pericarditis must remain unsettled until the coming of an intra-pericardial effusion, which, however, is usually not long delayed.

Now, upon the advent of fluid, the apex-beat



gets hidden, while pulsation is transferred to another part of the ventricle, and soon this, too, is lost in the increasing accumulation. If the sac fills full, there may be more or less bulging over the heart, and the intercostal spaces may become widened and immovable.

Upon percussion there is flatness from below upward in the parasternal line and across the præcordial region, so that a pyramid is embraced within its borders, whose apex may reach to the second rib, and base extend as far as or farther than the nipple-lines.

Upon auscultation, the friction is confined to the base of the heart or has wholly disappeared, while over the apex the sounds of the heart are either weak or absent, and around the organ in the area of flatness the respiratory murmur is suppressed.

With the retreat of the fluid, flatness recedes, friction comes back to remain a varying period, and then fades away. At the same time the respiratory murmur returns to the portion of the lung that was compressed by the effusion, the apex-beat regains its position, and the sounds of the heart are gradually restored to their normal vigor.

Where adhesions follow in the wake of an effusion, there is supposed to be a sinking in of two or

three adjacent intercostal spaces over the ventricle, with its contractions; but there is scanty proof of the correctness of this supposition, and, accordingly, there is no absolute certainty in the diagnosis of such a condition.

#### HYDROPERICARDIUM.

A passive effusion of the pericardial sac, of course, will not be ushered in by friction nor attended by fever, but it is identical, in its physical manifestations of fluid, with pericarditis. Hence, the student must employ the tests already given for the stage of effusion, and carefully consider the history and symptoms, remembering that hydropericardium is very likely to be associated with dropsy in the body elsewhere.

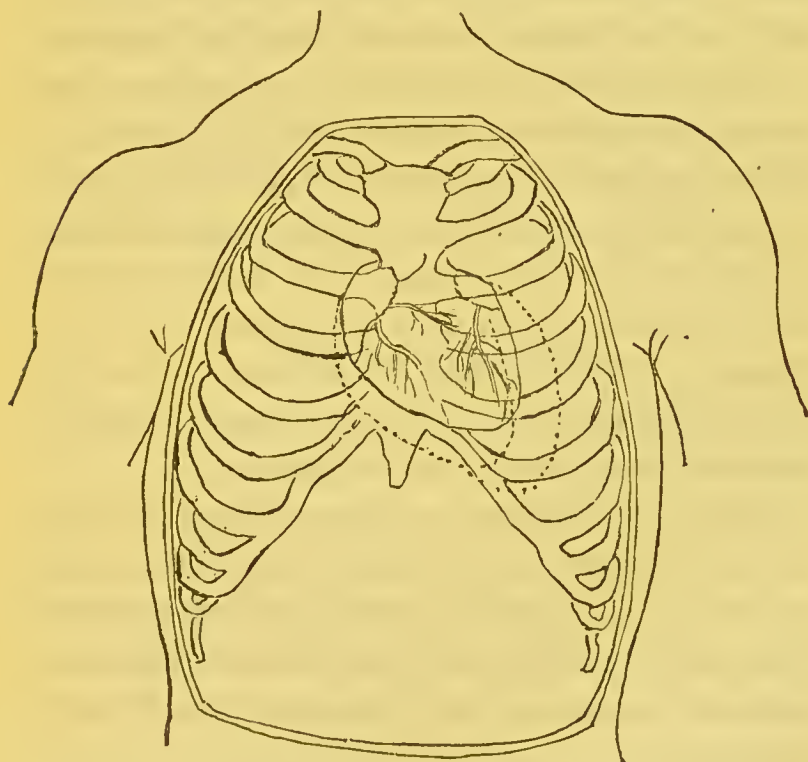
#### CARDIAC HYPERTROPHY.

The general term *hypertrophy*, as applied to the heart, includes two special forms, namely, an overgrowth of the muscles without dilatation of the cavities, which is simple hypertrophy; and a dilatation of the cavities, with an overgrowth of their walls, which is eccentric hypertrophy—and of these two varieties, the latter is the more common.

Upon inspection, in simple hypertrophy of the

heart, there is seen a heaving, regular impulse, and the apex is found below the fifth intercostal space, and beyond the left nipple-line.

In eccentric hypertrophy, the apex is carried downward, but a little further to the left than in simple hypertrophy, and, moreover, the impact is somewhat less forcible ; and, if the enlargement be



Diagram, dotted line to the left showing area of enlargement of left, dotted line to the right area of enlargement of the right, side of the heart.

confined to the right side, pulsation is most noticeable at the epigastrium, while the apex is pushed to the left.

With children, owing to the flexibility of the parietes, there is considerable bulging over the heart when it is enlarged.

By palpation, the force of the organ's stroke against the chest-wall, and the displacement of the apex, can be better appreciated than by inspection.

Upon percussion, the area of cardiac flatness and deep-seated dullness is extended in the direction of the augmentation of the heart. The dullness, which in health does not exceed the left nipple-line, goes beyond this limit in company with the left ventricle, and the flatness follows the receding lung in the same direction; and there will be dullness at the right of the sternum, if this side of the heart is enlarged, where it is otherwise not demonstrable.

In very thin persons an increase in the size of the left auricle may cause pulsation near the left edge of the sternum in the second or third intercostal space, and now and then, under these circumstances, dullness is carried up the parasternal line above the third rib.

Upon auscultation, in simple hypertrophy there is a dull, prolonged, thud-like first sound over the

apex, owing to a predominance of the muscular element in the sound, and often an accentuation of the second sound at the base, because of an increase in the blood-pressure.

With eccentric hypertrophy, however, the first sound is clear and loud, and the second less intense than in the former variety of enlargement.

When this increase of size is restricted to the left side of the heart, the mitral is plainer than the tricuspid, and the aortic than the pulmonary sound; and, likewise, when the augmentation is confined to the right side, the intensity is transferred to the tricuspid and pulmonary valves.

#### CARDIAC DILATATION.

It is commonly understood, by dilatation of the heart, that the chambers are enlarged, while, at the same time, their walls are reduced in thickness. More properly speaking, this is passive and eccentric hypertrophy is active dilatation.

There is no visible cardiac impulse with the affection, except in very thin persons, and even then it is indistinct, wide-spread, and wavy, instead of well-defined, concentrated, and steady, as in hypertrophy. This pulsation extends below but more especially

beyond the normal position, unless the enlargement be confined to the right side of the heart, when the impulse is mainly in the epigastric region.

If appreciable to the hand, the impact is weak and irregular, and in marked contrast with the forcible stroke of hypertrophy.

Upon percussion, the organ is found much increased in size, and particularly in its lateral dimensions.

Upon auscultation, there is revealed a clear, sharp, valvular first sound, because of the diminished muscular element to this sound, or else a very feeble one, owing to the impending asystolia. Moreover, there will frequently be a faint mitral systolic murmur, in lieu of the sound, from the resulting patency of the auriculo-ventricular valve.

But, then, it must be remembered that the impulse of the heart may be weak from general debility, and, on the other hand, strong simply from excitement; and, furthermore, displaced by extrinsic causes: hence, these possibilities should be carefully weighed before concluding that there is either hypertrophy or dilatation.



## FATTY DEGENERATION OF THE HEART.

The importance of detecting a fatty metamorphosis of the heart is always great, but at no time greater than when the administration of an anæsthetic becomes needful; for, though a stationary valvular lesion, or even a slowly progressive one that is well compensated, does not of itself invariably preclude the use of these agents, yet fatty degeneration, on the other hand, uniformly renders their employment extremely hazardous, at least for surgical purposes.

Now, while this fatty condition of the heart is not impossible in early life, it is much more liable to occur in persons well advanced in years. Thus, a patient past fifty, more commonly a man, with a pale, pasty skin, bedewed with clammy perspiration, who suffers from palpitation of the heart and dyspnœa, and that peculiar ascending and descending rhythm of breathing termed Cheyne-Stokes respiration, would furnish excellent reasons for a suspicion of fatty decay, and especially if he also had more or less angina pectoris, with now and then a pseudo-apoplectic seizure; and add to this atheroma of the arteries and a true arcus senilis, and the suspicions would pass into the realms of probability.

But the physical signs, aside from those of the organic lesions with which this disease may be associated, are somewhat meager. The impulse of the heart is either invisible, or very faintly distinguishable. There may be little if any enlargement, or the extreme expansion of dilatation. Upon auscultation the sounds of the heart are feeble, and the first sound at the apex is but a short flap of the leaves, or else it is lost.

Lastly, the action of the heart may be regular and abnormally slow, or rapid and very irregular, and withal so weak that many of the contractions fail to send an appreciable wave to the wrists.

Still, after all, it too often comes to pass that death takes place where neither signs nor symptoms had given warning of the mural decay found in the end to have been the cause of sudden dissolution.

#### AORTIC STENOSIS.

Since an obstruction at the aortic orifice puts an additional tax upon the left ventricle, and, at the same time, throws the blood into sonorous vibrations along the aorta, the muscles of this ventricle become hypertrophied, and a murmur is generated with every cardiac systole.

Consequently, the apex is seen, upon inspection, striking the chest below the fifth intercostal space, and somewhat beyond the left nipple-line; and, moreover, the impulse may be so strong as to cause a slight upheaving of the præcordia with each ventricular contraction.

By palpation, the force of this impact is appreciated, and also, in many instances, a systolic thrill over the base of the heart can be felt.

By percussion there is revealed an extension of the flatness and dullness of the heart to the left and downward.

Upon auscultation there is a bruit with greatest intensity close to the sternum in the second right intercostal space, with rhythm systolic, and with direction upward. It is usually harsh in quality, and sometimes both loud and harsh. The aortic second sound may be obliterated by this lesion. Now and then these murmurs are loudest at mid-sternum, on a level with the third costal cartilages, or to the left of this bone, in the second and possibly the third intercostal space; and they may be conveyed by conduction along the anterior surface of the chest to the left. Still, the only lesion that might, under these circumstances, be mistaken for aortic stenosis, is a contraction of the pulmonary orifice;

but this is extremely rare, and would be associated with hypertrophy of the right and not the left ventricle.

Neither mitral nor tricuspid systolic murmurs are carried very far upward, nor, as a rule, are they harsh in quality. A dynamic bruit may be excluded if the heart is tranquil, and the absence of anæmia would preclude a hæmic murmur; and, furthermore, a dynamic murmur, a hæmic bruit, or a simple roughening of the aortic orifice would not be attended by hypertrophy of the left ventricle.

The physical signs, then, of aortic stenosis are those of hypertrophy of the left ventricle, and of an aortic systolic murmur; and, though it is not impossible for this lesion to exist without a murmur, the obstruction could not be very extensive without producing an effect upon the left ventricle.

#### AORTIC INSUFFICIENCY.

From inadequacy of the aortic valve, blood is returned to the left ventricle, and eddies are formed in the reflux, so that the left ventricle becomes enlarged, and also diastolic bruits are produced.

Therefore, by inspection, the apex is located well to the left of, and below its normal position.

By palpation the impulse, though moderately strong, is less concentrated than in simple hypertrophy.

Upon percussion, the dullness of the heart is defined beyond the left nipple-line, and below the fifth intercostal space, and the flatness increases in proportion to the dullness.

Upon auscultation, there is a diastolic murmur, that may be loud or soft, and harsh or smooth, and which has its point of maximum intensity in the second right intercostal space near the sternum. This bruit extends up the arteries, and also down with the reflux, and it may be loudest at the lower end of the sternum. But sometimes it can be heard only at mid-sternum, on a level with the third costal cartilages, or at the left edge of this bone, a little lower.

If the aortic second sound is not wholly obscured by the murmur, it is an evidence that the valve is not entirely defective; whereas, if there is no valvular sound, and the murmur is soft and abrupt, there is probably free regurgitation; and the less opposition to the backward flow of the blood, the sooner the circulation of the heart, and thereby its nutrition, will suffer.

A diastolic bruit is the most constant of bruits,

and it is not likely to exist without regurgitation, nor, on the other hand, is regurgitation likely to occur without a murmur, but the insufficiency may be due to a dilatation of the aorta, and not necessarily to a lesion of the valve.

It is not often that the lines are clearly drawn between stenosis and patency of the aortic valve; and, where the lesions coexist, there are commonly two murmurs, one with systole and one with diastole. These are to-and-fro, oftentimes loud, harsh bruits, and widely diffused. Moreover, the heart is extensively enlarged by this combination.

The attempt has been made to formulate a method for deciding whether stenosis or patency predominates in a given lesion, by the relative quality, and also by the direction of the bruits; and, inasmuch as the prognosis is best where there is most obstruction, such a decision is desirable. But these distinctions are more or less fallacious, while, on the other hand, considerable reliance can be placed upon the nature of the radial pulse; for in extensive regurgitation this pulse is quite characteristic. A large volume of blood is thrown into the arteries and with great force by the hypertrophied left ventricle, and the wave strikes the wrist in a full and abrupt manner, but it falls away just as abruptly, owing to



the reflux into the left ventricle. This is known as the water-hammer, or as Corrigan's pulse, and it is made still more obvious by elevating the patient's arm. Now it follows that the more stenosis there is the more sustained will be the column of blood, and the less sudden the recoil from the wrist, and therefore the smaller the regurgitation.

To recapitulate, the physical signs of aortic insufficiency are those of enlargement of the left ventricle, and of an aortic diastolic murmur; while there are both systolic and diastolic murmurs, as well as an enlargement of the left ventricle, in stenosis with insufficiency.

#### MITRAL STENOSIS.

As a result of obstruction at the mitral aperture, vibrations are set up in the blood-stream as it is propelled into the left ventricle, and the left auricle and right ventricle become enlarged, while the left ventricle, in uncomplicated stenosis, is not only not enlarged but frequently decreased in size.

Upon inspection, pulsation is noticeable in the epigastrium, and, if the patient is very thin, also over the left auricle, in the vicinity of the sternum, at the third left intercostal space.

By palpation the apex is located about in its nor-

mal position ; and often over the left ventricle a thrill is communicated to the hand during the last portion of the second silence of the heart, that runs up to the apex-beat. The presence of this thrill, even in the absence of a bruit, strongly points to stenosis.

Upon auscultation, a harsh, grating, unusually loud murmur is heard just above the apex, where also it is most intense ; yet, however loud, it has a very limited area of diffusion, and is rarely if ever conveyed to the back. This bruit is auricular systolic, or, with respect to the ventricle, presystolic in rhythm ; that is to say, it occurs during the latter part of the second silence of the heart, while the auricle is contracting, or just before the systole of the left ventricle. The pulmonary second sound is intensified, and the degree of accentuation is thought to indicate the amount of the obstruction. On the other hand, there is so little stress upon the aortic second sound that it is almost inaudible at the mitral area. There may be also a reduplication of the cardiac second sound from a lack of simultaneous action of the two sides of the organ.

A mitral presystolic murmur, as a rule, is not difficult to recognize. It is lost as the stethoscope is moved far in any direction from the apex, and while listening, if the examiner will place a finger upon the

carotid artery, which beats in synchronism with the ventricle, to make sure of ventricular systole, he will hear a murmur coming up to the first sound and to apical impact that is suddenly cut off, as it were, in the midst of its fullness, by the first sound. There is some danger, however, of mistaking the first for the second sound, under these circumstances, owing to the altered quality of the former, and because the second sound may fail to reach the mitral area. But a knowledge of this possible source of error should enable the examiner to avoid being misled thereby.

It is seen, therefore, that the diagnosis of mitral stenosis rests upon the presence of a mitral presystolic bruit, a presystolic thrill, and upon the evidences of enlargement of the left auricle and right ventricle, but not of the left ventricle.

**Mitral Stenosis with Insufficiency.**—Stenosis with, at the same time, patency of the mitral orifice is attended by two murmurs, which in some instances remain separate and in others become continuous. The first murmur is presystolic and the second systolic in rhythm; and a very good way to distinguish one murmur from the other is by moving the stethoscope to the left, beyond the limit of presystolic diffusion, where the mitral systolic murmur still continues audible. With a double lesion, the left ven-

tricle is enlarged in proportion to the excess of patency over stenosis of the valve, and of hypertrophy over dilatation of the left auricle.

This is the commonest affection of the mitral valve, yet two murmurs are by no means the absolute rule. If the student will bear in mind the great muscular power of the left ventricle, compared with that of the left auricle, he will have no difficulty in understanding, especially if this auricle has passed into secondary dilatation, why a mitral systolic may be the only murmur, even when stenosis is the chief lesion; for the strong ventricle will drive the blood back through the patent orifice, and thus develop a systolic murmur, after the weak auricle has failed to throw the direct current into audible vibrations. These comprise many of the mitral systolic murmurs that are transmitted neither to the back nor far if at all to the left.

Now and then instances arise wherein two or three ineffectual attempts at ventricular systole occur before a well-defined murmur or a radial pulsation is perceptible—not in this case from an asystolia or weakness of the ventricle, but when there is reason to believe its contractility good, from (in the writer's opinion) a delay in the filling of that chamber, possibly in part through feebleness of the auricle, yet

mainly from the obstruction offered at the mitral valve to the auriculo-ventricular current.

Finally, it should be known that these two murmurs are inconstant and interchangeable as well, but, once established, the lesion is always present; from which it may be inferred that a difference of opinion between competent examiners, respecting the diagnosis on separate occasions, ought to be adjusted upon a basis of this understanding.

#### MITRAL INSUFFICIENCY.

If, during the systole of the left ventricle, blood is returned to the left auricle, a murmur will arise with each contraction, and the left auricle will become enlarged, then the right ventricle, and also to some degree the left ventricle.

Upon inspection, there is epigastric pulsation, and the apex is depressed and carried to the left.

By palpation, the impulse is found to be strong or weak in proportion to the hypertrophy or dilatation. Should the patient be very thin, the action of the enlarged left auricle would be felt; and, too, by palpation, a systolic thrill is sometimes appreciable over the left ventricle.

Upon percussion, an increase in the area of flat-

ness and dullness may be developed vertically as well as horizontally ; yet all percussion-signs of the heart depend somewhat upon thinness of the chest-wall for their utility.

By auscultation there is revealed a soft, blowing, sometimes musical bruit, whose greatest intensity is over the apex or mitral area, and that is systolic in rhythm. Taking the place, or part of the place, of the first sound at the apex, this murmur is diffused to the left, and also transmitted through to the back, where it is audible near the lower angle of the left scapula, and at times along the spine from the fourth to the eighth vertebra, and even to the right of the spinal column. But when insufficiency is complicated by stenosis, so that regurgitation is slight, or when ventricular systole is weak, the area of diffusion is circumscribed, and the murmur is inaudible at the back. Moreover, there is often considerable accentuation of the pulmonary second sound. The less the murmur encroaches upon the click of the mitral valve, the smaller the amount of the reflux.

A mitral is distinguished from a tricuspid systolic bruit by its point of maximum intensity and the direction which it takes. The former fades toward the right, whereas a tricuspid bruit grows plainer and is loudest at the lower end of the sternum.



It is possible for an emphysematous lung to obscure both mitral sounds and bruits, or, by depressing the apex, to carry them in the direction of the tricuspid region.

From the foregoing it is evident that a mitral systolic murmur, associated with enlargement of the left auricle and of each ventricle, is a proof of more or less reflux at the mitral orifice ; and the more completely the bruit invades the first sound at the mitral valve, and the greater the intensification of the pulmonary second sound, the more extensive the regurgitation.

#### TRICUSPID INSUFFICIENCY.

A reflux at the tricuspid orifice enlarges the right auricle, and if the patency of the valve, caused by either valvulitis or simple dilatation, be due primarily to mitral disease, which is commonly the case, there will be an enlargement not only of the right auricle, but also of the right ventricle, left auricle, and, with mitral insufficiency, of the whole heart. On the other hand, where the incompetency is owing to a chronic pulmonary disease only, as it sometimes is, the increase of size will be confined to the right side of the organ.

Upon inspection, there is seen an epigastric and

perhaps an auricular impulse ; while, if regurgitation be extensive, there is also a systolic pulsation in the right and, possibly, the left jugular vein.

On palpation, unless there be hypertrophy of the left ventricle, the apex-beat is either absent or at least not strong.

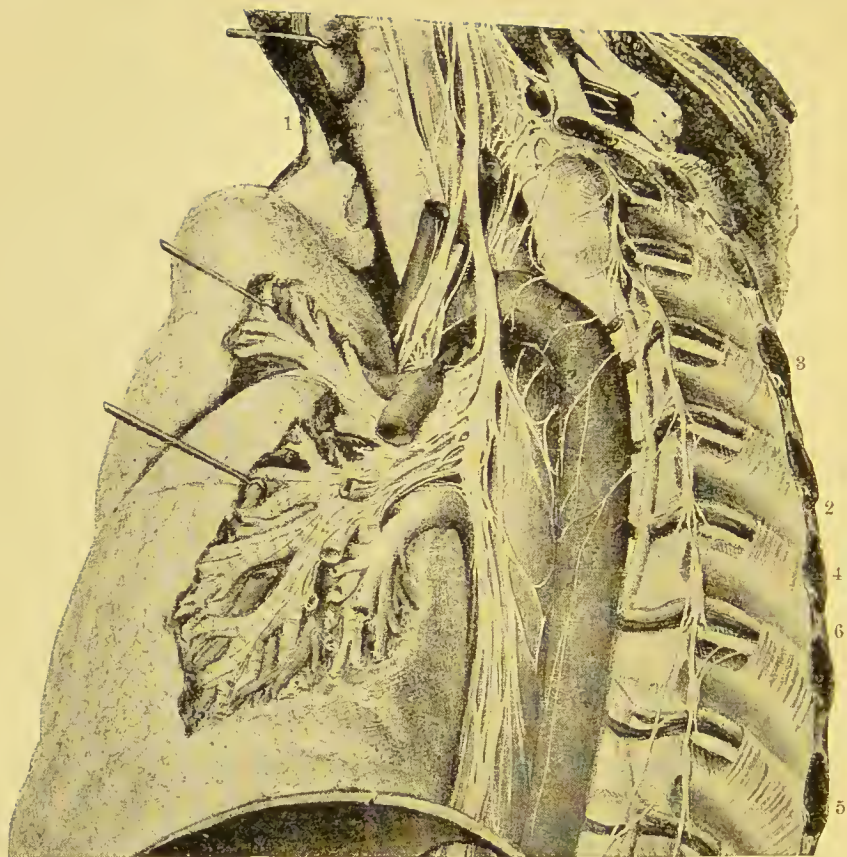
To decide between a transmitted impulse from the carotid artery and an original pulsation in the jugular vein, the examiner has but to stop the downward current of the vein by compressing this vessel at the middle of the neck ; for a simulated venous pulse ceases below the pressure, whereas the backward flow from the heart through the vein will continue.

By percussion there is developed an increase in the dullness over the heart, especially to the right of the sternum.

Upon auscultation, a soft, blowing, systolic murmur is revealed, that takes the place, or part of the place, of the first sound in the tricuspid area. This murmur is superficial, not widely diffused, and is seldom audible above the third rib.

Although a mitral systolic bruit may reach the tricuspid area, and thereby convey an erroneous impression that the right side of the heart is affected, yet, in such an event, the fact must be recalled that





THORACIC CAVITY.—LUNG DRAWN FORWARD. BRONCHI AND PULMONARY VESSELS EXPOSED. VIEW FROM THE LEFT SIDE (HIRSCHFELD).

- 1, trachea ; 2, œsophagus ; 3, arch of the aorta ; 4, left pneumogastric nerve ;  
5, thoracic aorta ; 6, intercostal vessels and nerves.

a tricuspid is scarcely ever propagated, like a mitral murmur, to the left beyond the apex ; and, too, under these circumstances, that the unaltered sounds of the tricuspid valve would be audible. And were an aortic systolic murmur to pass down the sternum, which as a rule it does not, it would be heard also in the carotids, where the tricuspid bruits never extend.

It follows, accordingly, that a tricuspid systolic murmur, with enlargement of the right side of the heart, indicates a reflux from the right ventricle to the auricle of the same side. And venous pulsation in the neck, together with cyanosis and œdema, would evince a very considerable regurgitation.

#### THORACIC ANEURISM.

It has been shown by careful investigation that the frequency of thoracic aneurism diminishes with the increased distance of the artery from the heart. Hence, of the three subdivisions of the aorta, the ascending is most commonly the seat of aneurism, the transverse next, while the descending portion is least commonly affected.

In the first instance, the tumor tends to the right of the sternum, in the vicinity of the second or third

costal cartilage, and it may eventually pass beyond the right nipple; in the second, it frequently does

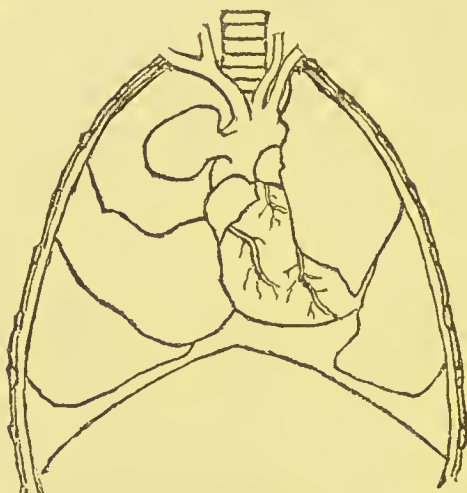


Diagram showing an aneurism of the ascending aorta.

not reach the surface of the chest, or at least project therefrom; still it may, now and then, be seen at the left of the sternum, near the second costal cartilage; in the third instance, the aneurism is usually deep-seated, and from its location invades the spinal column, rather than the anterior part of the thorax.

Upon inspection, if the tumor has involved the front wall of the chest, there will be bulging over this area, and more or less impairment in the respiratory play of the implicated region. Moreover, a pulsation is visible in this protuberance that keeps



time with the beating of the heart; and, even before there is a perceptible swelling, this impulse may be seen by looking across the chest, instead of by taking a front view. So, too, some of the results of pressure are found on inspection, such as fullness of the veins and œdema in the neck and upper extremities, caused by obstruction of the descending vena cava.

By palpation, a thrill is sometimes felt with the hand over the aneurism, and, when the tumor can be grasped, also an expansive impulse that is peculiar to this malady.

On mensuration, an increase in the interval between the median line and the nipple of the affected side is shown.

By percussion, let it be the gentlest of strokes, flatness is disclosed over that part of the tumor which impinges upon the chest-wall, and dullness, or possibly tympanitic resonance, over the compressed lung.

Through auscultation a murmur may be revealed that is usually single and systolic in rhythm, and which is not infrequently louder than most cardiac bruits; but often there will be nothing more than an impulsive throb and the intensified heart-sounds over the aneurism; and, should there happen to be

pressure upon a large bronchus, vesicular breathing would be suppressed, or at least enfeebled in the region cut off, and sonorous râles would become audible. Lastly, vocal resonance may be absent in this vicinity as well as over the tumor.

Since an aneurism seated in the transverse portion of the aorta is not always inclined toward the surface, fewer distinctive signs are apt to arise than when the first division of the vessel is occupied; but, on the other hand, the pressure symptoms are more numerous. For example, there is dyspnoea from compression of the trachea, the left bronchus, or else of the pneumogastric nerves. In the latter case the difficulty is intermittent, and there is a loud, dry, metallic cough; in the former conditions it is continuous, and the cough is attended by blood-tinged sputa. With obstruction of the air-tubes there will be sonorous respiration; pressure upon the pneumogastric nerve produces paralysis of the vocal cord of the affected side. This loss of function in the cord is clearly shown by laryngoscopic examination. Where the œsophagus is implicated, there is recurring dysphagia. Pressure upon an artery causes delay or, possibly, disappearance of pulsation in the distal extremity of the vessel; pressure upon the cervical sympathetic nerves is manifested by a con-

traction of the pupil in the eye of the side involved. An inequality of the pupils, however, is not necessarily a sign of disease, for it is sometimes found in healthy persons. Finally, pressure upon bone, cartilage, and nerve gives pain.

Wherefore, it may be concluded that a tumor of the anterior thoracic parietes, pulsating synchronously with the heart, outside its precincts, in coequal or greater force, that is expansile withal, and which is attended by some of the evidences of pressure, is undoubtedly an aneurism.



## I N D E X.

---

- Abscess of lung, 105.  
Absence of vesicular breathing, 68.  
Absorption (pleurisy), 118.  
Accentuation of heart-sounds, 154.  
Acoustics, 44.  
Adhesion (pleural), 119.  
    (pericardial), 175.  
Adventitious signs, 70.  
Ægophony, 79.  
Alterations in symmetry of chest,  
    21.  
Amphoric breathing, 67.  
    resonance, 48.  
    voice, 79.  
Anæmia, 20.  
Anæmic bruit, rhythm of, 162.  
    theory of, 155.  
Anæsthesia in heart-disease, 181.  
Aneurism (thoracic), 195.  
Aorta, thoracic, 142.  
Aortic insufficiency, 184.  
    stenosis, 182.  
Arteria innominata, 142.  
Artery, pulmonary, 142.  
Aspirator, 117.  
Asthma, 89.  
Attributes of sound, duration, 44.  
    Attributes of sound, intensity, 44.  
        pitch, 44.  
        quality, 44.  
        rhythm, 65.  
Auricular systolic murmurs, 165.  
Auscultation, definition of, 58.  
    directions for, 63.  
    immediate, 58.  
    in disease, 67.  
    in health, 64.  
    mediate, 58.  
    of heart, 151.  
    precautions in, 64.  
Auscultatory percussion, 53.  
Auscultatory signs, in disease, 67.  
    in health, 64.  
Auxiliaries to diagnosis, 18.  
Average temperature in health, 32.  
Axillary line, 9.  
Axillary region, in phthisis, 133.  
    in pleurisy, 114.  
Bacillus tuberculosis, 26.  
Binaural stethoscope, adjustment  
    of, 63.  
    selection of, 63.  
    value of, 61.

- Breathing, intensity of, 66.  
 Bronchial breathing, 66.  
   puff, 103.  
 Bronchiectasis, 85.  
 Bronchitis, acute, 82.  
   capillary, 86.  
   chronic, 84.  
   croupous, 88.  
 Broncho-cavernous breathing, 133.  
 Broncho-vesicular breathing, 66.  
 Bronchophony, 79.  
 Bubbling râles, 74.  
 Bulging and expansion of chest, 22.  
 Calorimetation, 31.  
 Cancer of the lung, 137.  
 Capillary bronchitis, acute, 86.  
 Cardiac dilatation, 179.  
   hypertrophy, 176.  
 Catarrhal bronchitis, acute, 82.  
 Cavernous breathing, 67.  
   whisper, 79.  
 Chest, description of, 9.  
 Chest-marks, 9.  
 Cheyne-Stokes respiration, 181.  
 Chronic catarrhal bronchitis, 84.  
   phthisis, 126.  
 Circular measurement of chest, 30.  
   in emphysema, 31.  
 Click, mucous, 75.  
 Clubbed finger-tips, 20.  
 Cog-wheel respiration, 68.  
 Compensatory hypertrophy, 157, 172.  
 Coughing, bronchial breathing by, 102.  
   bronchial puff by, 103.  
   friction by, 108.  
 Coughing, gurgles by, 133.  
   vesicular breathing by, 83.  
 Corrigan's pulse, 187.  
 Cosmetic pencil, 9.  
 Cracked-pot sound, 48.  
 Crepitant râles, 73.  
 Croupous bronchitis, 88.  
 Depression and retraction of chest, 22.  
 Diagnosis of diseases of lungs, 82.  
 Diastolic murmurs, 163.  
 Dilatation of bronchi, 85.  
   of heart, 179.  
   sounds in, 153.  
 Diminished breathing, 67.  
 Divided respiration, 68.  
 Dry pleurisy, 107.  
 Dual function of the ears, 61.  
 Dullness, 47.  
 Duration of sound, 44.  
 Emphysema, pulmonary, 93.  
 Empyema or pyothorax, 116.  
 Endocarditis, 170.  
 Exaggerated breathing, 67.  
 Expectoration of, acute bronchitis, 25.  
   cancer of lungs, 26.  
   capillary bronchitis, 25.  
   chronic bronchitis, 25.  
   fibrous bronchitis, 26.  
   hæmoptysis, 25.  
   œdema of lungs, 26.  
   phthisis, 26.  
   pneumonia, 27.  
 Expiration, absence of, 66.  
   prolonged, 68.



Exploration of the heart, 140.

Extraneous sounds, 77.

Fatty degeneration of heart, 181.

Fibroid phthisis, 135.

Fibrinous exudation (pleurisy), 107.

Flatness, 47.

Friction, 76.

fremitus, 29.

Gangrene, pulmonary, 136.

Gurgling râles, 75.

Hæmoptysis, 91.

Half-circumference of chest, 30.

Harsh breathing, 71.

Heart, abnormal sounds of, 153.

analysis of sounds of, 151.

apex of, 147.

auscultation of, 151.

compensation of, 157.

diagnosis of diseases of, 170.

effect of valvular disease on,  
156.

emphysema masking sounds of,  
153.

flatness of, 150.

function of, 145.

inspection of, 146.

mensuration of, 149.

methods of examining, 146.

murmurs of, 154.

normal sounds of, 143.

palpation of, 149.

percussion of, 149.

position of, 140.

relative site of valves of, 142.

superficial region of, 140.

thermometry in disease of, 149.

Heart, timing the, 152.

vocal resonance over, 152.

Heart-murmurs, 154.

anæmic, 162.

associated, 167.

curable, 163.

determination of, 160.

diastolic, 163.

dormant, 159.

dynamical, 162.

exceptional positions of, 164.

genesis of, 158.

illustration of, 155.

possible, 159.

presystolic, 165.

probable, 159.

quality of, 157.

spurious, 159.

systolic, 159.

temporary tricuspid, 163.

theory of, 155.

transient, 171.

Hydropericardium, 176.

Hydrothorax, 121.

Hypertrophy of heart, 176.

sounds in, 153.

compensatory, 172, 157.

Immediate percussion, 41.

Inspection, 13.

of heart, 146.

Intensity of sound, 44.

Interlobular fissures, 12.

Interrupted respiration, 68.

Interstitial pneumonia, 135.

Inspiration, 66.

Inspiratory expansion of chest, 30.

Key-note of resonance, 47.

- Lobular pneumonia, 105.  
Localized depressions of chest, 23.  
Location of kidneys, 16.  
    liver, 13.  
    lungs, 10.  
    spleen, 15.  
    stomach, 14.  
  
Mammillary line, 9.  
Mechanism of heart-sounds, 145.  
Mediate percussion, 41.  
Membranous casts of bronchi, 26.  
Mensuration, 30.  
Metallic tinkle, 75.  
Methods of examining the heart, 146.  
Miliary tuberculosis, acute, 134.  
Mitral insufficiency, 191.  
Mitral stenosis, 187.  
    with insufficiency, 189.  
Modification of heart-sounds, 153.  
Movements of thorax, in disease, 24.  
    in health, 23.  
Myoidema, 127.  
  
Œdema, pulmonary, 96.  
  
Palpation, 28.  
Parasternal line, 9.  
Pectoriloquy, 79.  
Percussion, 40.  
    auscultatory, 53.  
    definition of, 41.  
    difficulty in, 44.  
    in disease, 55.  
    in health, 49.  
    manipulations of, 43.  
    of heart, 149.  
  
Percussion, positions for, 42.  
    respiratory, 54.  
    rules for, 44.  
Pericardial murmurs, 166.  
Pericarditis, 173.  
Phthisis pulmonalis, 125.  
    acute, 125.  
Pigeon-breast, 22.  
Pitch of sound, 44.  
Pleurisy, 106.  
    acute, 107.  
    curvilinear flatness in, 111.  
Plexor and pleximeter, 41.  
Pneumo-hydrothorax, 121.  
Pneumonia, lobar, 98.  
    lobular, 105.  
Pneumo-pyothorax, 121.  
Pneumothorax, 121.  
Position for listening to heart, 144.  
Presystolic murmurs, 165.  
Progressive valvular lesions, 172.  
Prolonged expiration, 68.  
Puerile respiration, 66.  
Pulmonary breathing, 65.  
    resonance, 47.  
Purulent effusion (pleurisy), 116.  
    infiltration of lung, 105.  
Pyothorax, 116.  
  
Quality of sound, 44.  
  
Râles, bubbling, 74.  
    crepitant, 73.  
    gurgling, 75.  
    sibilant, 71.  
    sonorous, 71.  
    splashing, 75.  
    subcrepitant, 73.  
    tracheal, 75.

## Recapitulation of:

- adventitious signs, 77.
- attributes of sound, 46.
- breathing, 70.
- resonance, 57.
- rhythm, 70.
- vocal resonance, 80.

Regional percussion in health, 48.

Relative site of the valves, 142.

Resistance, sense of, in percussion, 42.

Resonance, 46.

modified by age, 49.

respiration, 44.

sex, 49.

Respiratory percussion, 54.

play of the lung, 51.

Rhonchal fremitus, 29.

Rhythm, 65.

Rusty sputa, 27.

Scapular line, 10.

Senile respiration, 66.

Serous effusion, 109.

Sibilant breathing, 71.

Sonorous breathing, 71.

Sound-waves, in pneumonia, 69.

in pleurisy, 113.

Splashing râles, 75.

Sputa, 25.

Static lesions of valves, 172.

Stethoscopes, 59.

Subacute pleurisy, 109.

Subercipitant râles, 73.

Succussion, 75.

## Summary; signs of:

- air and fluid in pleural sac, 124.
- asthma, 91.

## Summary; signs of:

- bronchitis, 85.
- capillary bronchitis, 88.
- chronic phthisis, 134.
- emphysema, 96.
- œdema (pulmonary), 97.
- pneumonia, 105.
- subacute pleurisy contrasted with pneumonia, 120.

Systolic murmurs, 159.

Table of temperatures, 40.

## Temperature in:

- acute miliary tuberculosis, 38.
- asthma, 39.
- bronchitis, 36.
- cancer of lung, 39.
- emphysema, 39.
- endocarditis, 39.
- hæmoptysis, 38.
- heart-disease, 39.
- hydrothorax, 39.
- œdema, 39.
- pericarditis, 39.
- phthisis, 38.
- pleurisy, 37.
- pneumonia, 37.

Temperature, significance of, 33.

Thermometer, manipulation of, 34.

Thermometry (see Calorimetry), 31.

Thoracentesis, 117.

Tracheal râles, 75.

Tricuspid insufficiency, 193.

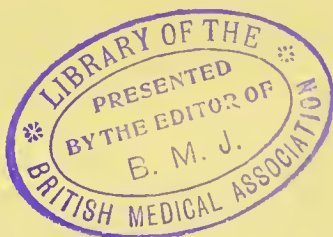
Trocar and cannula, 118.

Tubular breathing (see Bronchial), 66.

Tympanic resonance, 48.

- |   |   |
|---|---|
| Ulcerative endocarditis, acute, 171.          | Vesiculo-tympanitic resonance, 48.<br>in pleurisy, 112. |
| Valves of the veins, 168.                     | Vocal fremitus, 28.                                     |
| Venous murmur or hum, 167.<br>theory of, 169. | resonance, 78.  |
| Vesicular breathing, 65.<br>resonance, 47.    | Water-hammer pulse, 187.                                |
|   | Whispering pectoriloquy, 79.                            |

THE END.



March, 1888.

SELECTED LIST  
OF  
NEW AND RECENT WORKS  
PUBLISHED BY  
H. K. LEWIS,  
136 GOWER STREET, LONDON, W.C.

---

\* \* For full list of works in Medicine and Surgery published by  
H. K. Lewis see Complete Catalogue sent post free on application.

---

ADOLF STRÜMPPELL, M.D.

Director of the Medical Clinic in the University of Erlangen.

**A TEXT-BOOK OF MEDICINE FOR STUDENTS  
AND PRACTITIONERS.** Translated from the latest  
German Edition by Dr. H. F. VICKERY and Dr. P. C. KNAPP,  
with Editorial Notes by Dr. F. C. SHATTUCK, Visiting Physician  
to the Massachusetts General Hospital, etc. Complete in one  
volume, with 111 Illustrations, cloth, 28s. [*Just published.*]

"It is to be hoped that this work may prove useful to practitioners and students alike. It has achieved great success in Germany, having very rapidly reached a third edition, and has been adopted as the text-book in the Theory and Practice of Medicine in the Medical Department of Harvard University." *Editor's Preface.*

"Prof. Strümpell, lately appointed Director of the Medical Clinic in the University of Erlangen, is well-known as one of the ablest of modern German physicians. His system of medicine occupies the same place in Germany at the present day as did the great work of Niemeyer ten or fifteen years ago."—*Canada Medical and Surgical Journal.*

"Of the German text-books of practice that have been translated into English, Professor Strümpell's will probably take the highest rank. Between its covers will be found a very complete and systematic description of all the diseases which are classed under the head of internal medicine . . . . It is one of the most valuable works of practice that we have, and one which every studious practitioner should have upon his shelves."—*New York Medical Journal.*

---

DRS. BOURNEVILLE AND BRICON.

**MANUAL OF HYPODERMIC MEDICATION.** Translated from the Second Edition, and Edited, with Therapeutic Index of Diseases, by ANDREW S. CURRIE, M.D. Edin., etc. Crown 8vo, 6s. [*Now ready.*]

ALFRED COOPER, F.R.C.S.

Surgeon to the St. Mark's Hospital for Fistula and other Diseases of the Rectum.

**A PRACTICAL TREATISE ON DISEASES OF THE RECTUM.** Crown 8vo, 4s. [Just published.]

H. CHARLTON BASTIAN, M.A., M.D., F.R.S.

Examiner in Medicine at the Royal College of Physicians; Physician to University College Hospital, and to the National Hospital for the Paralysed and Epileptic; etc.

**PARALYSES: CEREBRAL, BULBAR, AND SPINAL.** A Manual of Diagnosis for Students and Practitioners. With numerous Illustrations, 8vo, 12s. 6d.

"One great feature of the book is the number of carefully and usefully arranged tables of diagnosis. These are eminently practical, and give the required knowledge in a nutshell, so that the hard-worked student can get his food in a concentrated form, and the busy practitioner can keep himself from rusting, or on emergency refresh his failing memory . . . We can as thoroughly recommend as we heartily welcome this book."—*Journal of Mental Science*.

"As a special work on the diagnosis or localization of a paralyzing lesion, we do not know of its equal in any language."—*Virginia Medical Monthly*.

FREDERICK T. ROBERTS, M.D., B.SC., F.R.C.P.

Examiner in Medicine at the Royal College of Surgeons; Professor of Therapeutics in University College; Physician to University College Hospital; Physician to the Brompton Consumption Hospital, &c.

**A HANDBOOK OF THE THEORY AND PRACTICE OF MEDICINE.** Sixth Edition, with Illustrations, in one volume of over 1000 pages, large 8vo, 21s.

"We heartily commend this handbook, not only to gentlemen preparing for the medical profession, but to those who have finished their professional education; as this work contains, in a brief and concise shape, all that the busy practitioner needs to know to enable him to carry on his practice with comfort to himself and with advantage to his patients."—*British Medical Journal*.

"We have already on more than one occasion expressed a high opinion as to the merits of this work. . . . From our experience of the 'Handbook,' we believe that it will always be popular amongst medical students, and that it is sufficiently classical to deserve a place on the bookshelves of every physician. . . . We heartily commend it as a reliable guide not less to the practical than to the theoretical study of medicine."—*Dublin Journal of Medical Science*.

BY THE SAME AUTHOR.

**THE OFFICIAL MATERIA MEDICA.** Second Edit., entirely rewritten in accordance with the latest British Pharmacopœia, fcap. 8vo, 7s. 6d. [Just published.]

"In our number for January, 1885, we noticed the first edition of Dr. Roberts' book, remarking that 'with this compendious book in his hand the student will have a safe and excellent guide to the official materia medica'. We say that yet more strongly of the present edition. It is essentially a book for students who, in the words of the author, will find the subject presented from every aspect which can fairly be required at examinations."—*Liverpool Medico-Chirurgical Journal*.



SIR WILLIAM AITKEN, KNT., M.D., F.R.S.

ON THE ANIMAL ALKALOIDS, THE PTOMAINES,  
LEUCOMAINES, AND EXTRACTIVES IN THEIR  
PATHOLOGICAL RELATIONS. Crown 8vo, 2s. 6d.

---

DR. R. ULZMANN.

ON STERILITY AND IMPOTENCE IN MAN. Trans-  
lated from the German with notes and additions by ARTHUR  
COOPER, L.R.C.P., M.R.C.S., Surgeon to the Westminster  
General Dispensary. With Illustrations, fcap. 8vo, 2s. 6d.

[Now ready.]

---

ANGEL MONEY, M.D., M.R.C.P.

Assistant Physician to the Hospital for Children, Great Ormond Street;  
and to the Victoria Park Chest Hospital.

TREATMENT OF DISEASE IN CHILDREN: In-  
cluding the Outlines of Diagnosis and the chief Pathologi-  
cal differences between Children and Adults. Crown 8vo,  
10s. 6d.  
*Lewis's Practical Series.* [Ready.]

"The work presents the charm of originality. The author has expressed his own idea, in his own way. . . . Still as its title implies, the book is chiefly concerned with 'treatment,' and the information given here is full and complete."—*Practitioner*.

"Of Dr. Money's work, which is essentially one on therapeutics, we can also speak highly. It gives a pretty complete exposition of the various methods of treatment which have been recommended by different authorities, and embodies the results of the author's experience at the Great Ormond Street Hospital. . . . His own recommendations are judicious and will meet with general approval."—*Lancet*.

---

FRANCIS HENRY CHAMPNEYS, M.A., M.B. OXON., F.R.C.P.  
Obstetric Physician and Lecturer on Obstetric Medicine at St. George's Hos-  
pital; Examiner in Obstetric Medicine in the University of London, etc.

EXPERIMENTAL RESEARCHES IN ARTIFICIAL  
RESPIRATION IN STILLBORN CHILDREN, AND  
ALLIED SUBJECTS. Crown 8vo, 3s. 6d.

---

HENRY DAVIS, M.R.C.S. ENG.

Teacher and Administrator of Anæsthetics to St. Mary's and the National  
Dental Hospitals.

GUIDE TO THE ADMINISTRATION OF ANÆS-  
THETICS. Fcap. 8vo, 2s.

BERKELEY HILL, M.B. LOND., F.R.C.S.

Professor of Clinical Surgery in University College; Surgeon to University College Hospital, and to the Lock Hospital.

AND

ARTHUR COOPER, L.R.C.P., M.R.C.S.

Surgeon to the Westminster General Dispensary, &c.

I.

**SYPHILIS AND LOCAL CONTAGIOUS DISORDERS.** Second Edition, entirely re-written, royal 8vo, 18s.

II.

**THE STUDENT'S MANUAL OF VENEREAL DISEASES.** Being a Concise Description of those Affections and of their Treatment. Fourth Edition, post 8vo, 2s. 6d.

SYDNEY RINGER, M.D., F.R.S.

Professor of the Principles and Practice of Medicine in University College; Physician to, and Professor of Clinical Medicine in, University College Hospital.

**A HANDBOOK OF THERAPEUTICS.** Eleventh Edition, revised, 8vo, 15s.

"...The work supplies a felt want, giving useful information which can be obtained from no other book, and which is of the utmost value in practice... The work has now become almost indispensable both to students of medicine and to practitioners."—*Practitioner*

"It is unquestionably the ablest work on Therapeutics which we possess in our language, and one which should be carefully perused not only by students, but also by practitioners....Ringer's *Therapeutics* is so well-known that it needs no commendation from us to ensure it a wide circulation."—*Edinburgh Medical Journal*.

R. DOUGLAS POWELL, M.D., F.R.C.P., M.R.C.S.

Physician to the Hospital for Consumption and Diseases of the Chest at Brompton, Physician to the Middlesex Hospital.

**DISEASES OF THE LUNGS AND PLEURÆ INCLUDING CONSUMPTION.** Third Edition, re-written and enlarged, with coloured plates and wood-engravings, 8vo, 16s.

"We commend this book as one which should be in the hands of every practitioner. It is plainly the outcome of wide experience, and it has been written in a thoughtful and practical manner, so that no one who studies its pages can fail to derive therefrom much that will stand him in good stead at the bedside."—*Lancet*.

"The present edition will take a high place in the estimation of practical physicians. Over and beyond the wide knowledge displayed and the judicial temper with which disputed points are discussed, there remains a striking characteristic of the book, which may perhaps be best called its helpfulness; difficulties in practice are honestly stated and sound practical advice is given, to the exclusion of vague generalisation or hearsay recommendations of new nostrums."—*British Medical Journal*.

ROBSON ROOSE, M.D., F.R.C.P. EDIN.

**GOUT, AND ITS RELATIONS TO DISEASES OF THE LIVER AND KIDNEYS.** Fifth Edition, crown 8vo, 3s. 6d. *[Just published.]*

---

C. W. MANSELL MOULLIN, M.A., M.D. OXON., F.R.C.S. ENG.  
Assistant Surgeon and Senior Demonstrator of Anatomy at the London Hospital; formerly Radcliffe Travelling Fellow and Fellow of Pembroke College, Oxford.

**SPRAINS; THEIR CONSEQUENCES AND TREATMENT.** Crown 8vo, 5s. *[Now ready.]*

---

LEWIS A. STIMSON, B.A., M.D.

Surgeon to the Presbyterian and Bellevue Hospitals; Professor of Clinical Surgery in the Medical Faculty of the University of the City of New York, etc.

**A MANUAL OF OPERATIVE SURGERY.** With three hundred and forty-two Illustrations. Second Edition, post 8vo, 10s. 6d.

---

HENRY R. SWANZY, A.M., M.B., F.R.C.S.I.

Examiner in Ophthalmic Surgery at the Royal College of Surgeons, Ireland; Surgeon to the National Eye and Ear Infirmary, Dublin; Ophthalmic Surgeon to the Adelaide Hospital, Dublin; Formerly Assistant to the late Professor A. von Graefe, Berlin.

**A HANDBOOK OF DISEASES OF THE EYE AND THEIR TREATMENT.** Second Edition, Illustrated with Wood Engravings, Colour Tests, etc., large post 8vo. *[In preparation.]*

"This is an excellent textbook, written by a surgeon of large experience and a thorough knowledge of the literature of his subject."—*The Edinburgh Medical Journal.*

---

EDGAR M. CROOKSHANK, M.B. LOND., F.R.M.S.

Professor of Bacteriology, King's College, London.

I.

**MANUAL OF BACTERIOLOGY: BEING AN INTRODUCTION TO PRACTICAL BACTERIOLOGY.** Illustrated with coloured plates from original drawings and numerous coloured illustrations embodied in the text. Second Edition, 8vo, 21s. *[Now ready.]*

II.

**PHOTOGRAPHY OF BACTERIA** Illustrated with 86 Photographs reproduced in autotype and numerous wood engravings, royal 8vo, 12s. 6d. *[Now ready.]*

BERKELEY HILL, M.B. LOND., F.R.C.S.

Professor of Clinical Surgery in University College; Surgeon to University College Hospital, and to the Lock Hospital.

**THE ESSENTIALS OF BANDAGING.** For Managing Fractures and Dislocations; for administering Ether and Chloroform; and for using other Surgical Apparatus. Sixth Edition, with Illustrations, fcap. 8vo, 5s.

---

CHARLES CREIGHTON, M.D.

I.

**ILLUSTRATIONS OF UNCONSCIOUS MEMORY**  
IN DISEASE, including a Theory of Alteratives. Post 8vo, 6s.

II.

**CONTRIBUTIONS TO THE PHYSIOLOGY AND**  
PATHOLOGY OF THE BREAST AND LYMPHATIC GLANDS. Second Edition, with wood-cuts and plate, 8vo, 9s.

III.

**BOVINE TUBERCULOSIS IN MAN:** An Account of the Pathology of Suspected Cases. With Chromo-lithographs and other Illustrations, 8vo, 8s. 6d.

---

W. H. O. SANKEY, M.D. LOND., F.R.C.P.

Late Lecturer on Mental Diseases, University College, and School of Medicine for Women, London.

**LECTURES ON MENTAL DISEASE.** Second Edition, with coloured plates, 8vo, 12s. 6d.

---

ROBERTS BARTHOLOW, M.A., M.D., LL.D.

Professor of Materia Medica and Therapeutics in the Jefferson Medical College of Philadelphia, etc.

I.

**A TREATISE ON THE PRACTICE OF MEDICINE**  
FOR THE USE OF STUDENTS AND PRACTITIONERS. With Illustrations, 5th Edition, large 8vo, 21s.

II.

**A PRACTICAL TREATISE ON MATERIA MEDICA**  
AND THERAPEUTICS. Sixth Edition, Revised and Enlarged, 8vo, 18s. [Just published.]

EDWARD WOAKES, M.D.

Senior Aural Surgeon and Lecturer on Aural Surgery at the London Hospital;  
Surgeon to the London Throat Hospital.

I.

**ON DEAFNESS, GIDDINESS, AND NOISES IN THE HEAD.**

**PART I.—POST-NASAL CATARRH, AND DISEASES OF THE NOSE CAUSING DEAFNESS.** With Illustrations, crown 8vo, 6s. 6d.

**PART II.—ON DEAFNESS, GIDDINESS, AND NOISES IN THE HEAD.** Third Edition, with Illustrations, crown 8vo. [*In preparation.*]

II.

**NASAL POLYPUS: WITH NEURALGIA, HAY-FEVER, AND ASTHMA, IN RELATION TO ETHMOIDITIS.** With Illustrations, crown 8vo, 4s. 6d. [*Now ready.*]

---

E. CRESSWELL BABER, M.B. LOND.

Surgeon to the Brighton and Sussex Throat and Ear Dispensary.

**A GUIDE TO THE EXAMINATION OF THE NOSE WITH REMARKS ON THE DIAGNOSIS OF DISEASES OF THE NASAL CAVITIES.** With Illustrations, small 8vo, 5s. 6d.

---

G. GRANVILLE BANTOCK, M.D., F.R.C.S. EDIN.

Surgeon to the Samaritan Free Hospital for Women and Children.

**ON THE USE AND ABUSE OF PESSARIES.** With Illustrations, Second Edition, 8vo, 5s.

---

W. BRUCE CLARKE, M.A., M.B. OXON., F.R.C.S.

Assistant Surgeon to, and Senior Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital; Surgeon to the West London Hospital; Examiner in Anatomy to the University of Oxford.

**THE DIAGNOSIS AND TREATMENT OF DISEASES OF THE KIDNEY AMENABLE TO DIRECT SURGICAL INTERFERENCE.** Demy 8vo, with Illustrations, 7s. 6d.

"The book throughout is carefully and pleasantly written."—*Practitioner*.  
"It is readable and trustworthy."—*British Medical Journal*.



8      New and Recent Works published by

---

FANCOURT BARNES, M.D., M.R.C.P.

Physician to the Chelsea Hospital; Obstetric Physician to the Great Northern Hospital, &c.

**A GERMAN-ENGLISH DICTIONARY OF WORDS AND TERMS USED IN MEDICINE AND ITS COGNATE SCIENCES.** Square 12mo, Roxburgh binding, 9s.

---

ALFRED H. CARTER, M.D. LOND.

Member of the Royal College of Physicians; Physician to the Queen's Hospital, Birmingham; Examiner in Medicine for the University of Aberdeen, &c.

**ELEMENTS OF PRACTICAL MEDICINE.** Fourth Edition, crown 8vo, 9s.

---

P. GAZEAX.

Adjunct Professor in the Faculty of Medicine of Paris, &c.

AND

S. TARNIER.

Professor of Obstetrics and Diseases of Women and Children in the Faculty of Medicine of Paris.

**OBSTETRICS: THE THEORY AND PRACTICE;** including the Diseases of Pregnancy and Parturition, Obstetrical Operations, &c. Seventh Edition, edited and revised by ROBERT J. HESS, M.D., with twelve full-page plates, five being coloured, and 165 wood-engravings, 1081 pages, royal 8vo, 35s.

---

W. H. CORFIELD, M.A., M.D. OXON.

Professor of Hygiene and Public Health in University College, London.

**DWELLING HOUSES:** their Sanitary Construction and Arrangements. Second Edition, with Illustrations, crown 8vo, 3s. 6d.

---

EDWARD COTTERELL, M.R.C.S. ENG., L.R.C.P. LOND.

Late House Surgeon, University College Hospital; Atkinson Morley Surgical Scholar, University College, London, etc.

**ON SOME COMMON INJURIES TO LIMBS:** their Treatment and After-Treatment including Bone-Setting (so-called). Imp. 16mo, with Illustrations, 3s. 6d.



WM. JAPP SINCLAIR, M.A., M.D.

Hon. Physician to the Manchester Southern Hospital for Women and  
Children and Manchester Maternity Hospital.

ON GONORRHEAL INFECTION IN WOMEN.  
Post 8vo. *[In the press.]*

---

J. MAGEE FINNEY, M.D. DUBLIN.

King's Professor of Practice of Medicine in School of Physic, Ireland;  
Clinical Physician to Sir Patrick Dun's Hospital.

NOTES ON THE PHYSICAL DIAGNOSIS OF LUNG  
DISEASES. 32mo, 1s. 6d.

---

J. MILNER FOTHERGILL, M.D.

Member of the Royal College of Physicians of London; Physician to the City  
of London Hospital for Diseases of the Chest, Victoria Park, &c.

I.

A MANUAL OF DIETETICS. Large 8vo, 10s. 6d.

II.

THE HEART AND ITS DISEASES, WITH THEIR  
TREATMENT; INCLUDING THE GOUTY HEART.  
Second Edition, entirely re-written, copiously illustrated with  
woodcuts and lithographic plates. 8vo, 16s.

III.

INDIGESTION, BILIOUSNESS, AND GOUT IN ITS  
PROTEAN ASPECTS.

PART I.—INDIGESTION AND BILIOUSNESS. Second  
Edition, post 8vo, 7s. 6d. *[Just published.]*

PART II.—GOUT IN ITS PROTEAN ASPECTS.  
Post 8vo, 7s. 6d.

---

ALFRED W. GERRARD, F.C.S.

Pharmaceutical Chemist; Examiner to the Pharmaceutical Society; Teacher  
of Pharmacy and Demonstrator of Materia Medica to University  
College Hospital, etc.

ELEMENTS OF MATERIA MEDICA AND PHAR-  
MACY. Crown 8vo, 8s. 6d.

---

LEWIS'S POCKET CASE BOOK FOR PRACTI-  
TIONERS AND STUDENTS. Designed by A. T.  
BRAND, M.D. Roan, with pencil, 3s. 6d. *nett.*

LEWIS'S PRACTICAL SERIES.

Under this title Mr. Lewis is publishing a complete series of Monographs embracing the various branches of Medicine and Surgery.

The volumes, written by well-known Hospital Physicians and Surgeons recognised as authorities in the subjects of which they treat, are in active preparation. The works are intended to be of a thoroughly Practical nature, calculated to meet the requirements of the general Practitioner, and to present the most recent information in a compact and readable form; the volumes will be handsomely got up and issued at low prices, varying with the size of the works.

THE FOLLOWING ARE NOW READY.

**TREATMENT OF DISEASE IN CHILDREN: INCLUDING THE OUTLINES OF DIAGNOSIS AND THE CHIEF PATHOLOGICAL DIFFERENCES BETWEEN CHILDREN AND ADULTS.** By ANGEL MONEY, M.D., M.R.C.P., Assistant Physician to the Hospital for Children, Great Ormond Street, and to University College Hospital. Crown 8vo, 10s. 6d. [Ready.

**ON FEVERS: THEIR HISTORY, ETIOLOGY, DIAGNOSIS, PROGNOSIS, AND TREATMENT.** By ALEXANDER COLLIE, M.D. (Aberdeen), Member of the Royal College of Physicians of London; Medical Superintendent of the Eastern Hospitals; Secretary of the Epidemiological Society for Germany and Russia. Coloured plates, cr. 8vo, 8s. 6d.

**HANDBOOK OF DISEASES OF THE EAR FOR THE USE OF STUDENTS AND PRACTITIONERS.** By URBAN PRITCHARD, M.D. (Edin.), F.R.C.S. (Eng.), Professor of Aural Surgery at King's College, London; Aural Surgeon to King's College Hospital; Senior Surgeon to the Royal Ear Hospital. With Illustrations, crown 8vo, 4s. 6d.

**A PRACTICAL TREATISE ON DISEASES OF THE KIDNEYS AND URINARY DERANGEMENTS.** By C. H. RALFE, M.A., M.D. Cantab., F.R.C.P. Lond., Assistant Physician to the London Hospital, late Senior Physician to the Seamen's Hospital, Greenwich. With Illustrations, crown 8vo, 10s. 6d.

**DENTAL SURGERY FOR GENERAL PRACTITIONERS AND STUDENTS OF MEDICINE.** By ASHLEY W. BARRETT, M.B. Lond., M.R.C.S., L.D.S., Dental Surgeon to, and Lecturer on Dental Surgery and Pathology in the Medical School of, the London Hospital. With Illustrations, crown 8vo, 3s.

**BODILY DEFORMITIES AND THEIR TREATMENT: A Handbook of Practical Orthopædics.** By H. A. REEVES, F.R.C.S. Ed., Senior Assistant Surgeon and Teacher of Practical Surgery at the London Hospital; Surgeon to the Royal Orthopædic Hospital, etc. With numerous Illustrations, crown 8vo, 8s. 6d.

\*.\* Prospectus of the Series, with Specimen pages, etc., post free on application.

---

**L**EWIS'S POCKET MEDICAL VOCABULARY.  
Over 200 pp., 32mo, limp roan, 3s. 6d.

HENEAGE GIBBES, M.D.

Lecturer on Physiology and on Normal and Morbid Histology in the Medical School of Westminster Hospital; etc.

**PRACTICAL HISTOLOGY AND PATHOLOGY.**

Third Edition, revised and enlarged, crown 8vo, 6s.

---

J. B. GRESSWELL, M.R.C.V.S.

Provincial Veterinary Surgeon to the Royal Agricultural Society.

**VETERINARY PHARMACOLOGY AND THERAPEUTICS.** Fcap. 8vo, 5s.

---

J. WICKHAM LEGG, F.R.C.P.

Assistant Physician to Saint Bartholomew's Hospital, and Lecturer on Pathological Anatomy in the Medical School.

I.

**ON THE BILE, JAUNDICE, AND BILIOUS DISEASES.** With Illustrations in chromo-lithography, 719 pages, roy. 8vo, 25s.

II.

**A GUIDE TO THE EXAMINATION OF THE URINE;** intended chiefly for Clinical Clerks and Students. Sixth Edition, revised and enlarged, with additional Illustrations, fcap. 8vo, 2s 6d.

---

WILLIAM THOMPSON LUSK, A.M., M.D.

Professor of Obstetrics and Diseases of Women in the Bellevue Hospital Medical College, &c.

**THE SCIENCE AND ART OF MIDWIFERY.** Third Edition, revised and enlarged, with numerous Illustrations, 8vo, 18s.

---

PATRICK MANSON, M.D., C.M.

Amoy, China.

**THE FILARIA SANGUINIS HOMINIS AND CERTAIN NEW FORMS OF PARASITIC DISEASE IN INDIA, CHINA, AND WARM COUNTRIES.** Illustrated with Plates, Woodcuts, and Charts. Deny 8vo, 10s. 6d.

WILLIAM MARTINDALE, F.C.S.

Late Examiner of the Pharmaceutical Society, and late Teacher of Pharmacy  
and Demonstrator of Materia Medica at University College.

AND

W. WYNN WESTCOTT, M.B. LOND.

Deputy Coroner for Central Middlesex.

**THE EXTRA PHARMACOPŒIA** with the additions  
introduced into the British Pharmacopœia 1885; and  
Medical References, and a Therapeutic Index of Diseases and  
Symptoms. Fourth Edition, revised, limp roan, med. 24mo, 7s.

[Now ready.]

---

A. STANFORD MORTON, M.B., F.R.C.S. ED.

Senior Assistant Surgeon, Royal South London Ophthalmic Hospital.

**REFRACTION OF THE EYE:** Its Diagnosis, and the  
Correction of its Errors, with Chapter on Keratotomy.  
Third Edition. Small 8vo, 3s.

---

WILLIAM MURRELL, M.D., F.R.C.P.

Lecturer on Materia Medica and Therapeutics at Westminster Hospital  
Examiner in Materia Medica and Therapeutics in the University  
of Edinburgh, and to the Royal College of Physicians,  
London.

I.

**MESSAGE AS A MODE OF TREATMENT.** Third  
Edition, crown 8vo, 4s. 6d. [Just published.]

II.

**WHAT TO DO IN CASES OF POISONING.** Fifth  
Edition, royal 32mo, 3s. 6d.

---

G. OLIVER, M.D., M.R.C.P.

I.

**ON BEDSIDE URINE TESTING:** a Clinical Guide to  
the Observation of Urine in the course of Work. Third  
Edition, considerably enlarged, fcap. 8vo, 3s. 6d.

II.

**THE HARROGATE WATERS:** Data Chemical and  
Therapeutical, with notes on the Climate of Harrogate.  
Addressed to the Medical Profession. Crown 8vo, with Map of  
the Wells, 3s. 6d.

R. W. PARKER.

Surgeon to the East London Hospital for Women and Children and to the Grosvenor Hospital for Women and Children.

I.

**TRACHEOTOMY IN LARYNGEAL DIPHTHERIA;  
AFTER - TREATMENT AND COMPLICATIONS.**  
Second Edition, with Illustrations, 8vo, 5s.

II.

**CONGENITAL CLUB-FOOT: ITS NATURE AND  
TREATMENT.** With special reference to the subcutaneous division of Tarsal Ligaments. 8vo, 7s. 6d.

---

G. V. POORE, M.D., F.R.C.P.

Professor of Medical Jurisprudence, University College; Assistant Physician and Physician in charge of the Throat Department of University College Hospital.

**L**ECTURES ON THE PHYSICAL EXAMINATION  
OF THE MOUTH AND THROAT. With an appendix  
of Cases. 8vo, 3s. 6d.

---

CHARLES W. PURDY, M.D. (QUEEN'S UNIV.)

Professor of Genito-Urinary and Renal Diseases in the Chicago Polyclinic,  
etc., etc.

**B**RIGHT'S DISEASE AND THE ALLIED AFFEC-  
TIONS OF THE KIDNEYS. With Illustrations,  
large 8vo, 8s. 6d.

---

D. B. ST. JOHN ROOSA, M.A., M.D.

Professor of Diseases of the Eye and Ear in the University of the City of New York; Surgeon to the Manhattan Eye and Ear Hospital; Consulting Surgeon to the Brooklyn Eye and Ear Hospital, &c., &c.

**A** PRACTICAL TREATISE ON THE DISEASES OF  
THE EAR, including the Anatomy of the Organ. Sixth  
Edition, Illustrated by wood engravings and chromo-lithographs,  
large 8vo, 25s.

---

W. JULIUS MICKLE, M.D., M.R.C.P. LOND.

Medical Superintendent, Grove Hall Asylum, London, etc.

**G**ENERAL PARALYSIS OF THE INSANE, Second  
Edition, enlarged and rewritten, 8vo, 14s.



JOHN SAVORY.

Member of the Society of Apothecaries, London.

**A COMPENDIUM OF DOMESTIC MEDICINE AND  
COMPANION TO THE MEDICINE CHEST.** In-  
tended as a source of easy reference for Clergymen, Master  
Mariners, and Travellers; and for Families resident at a distance  
from professional assistance. Tenth Edition, fcap. 8vo, 5s.

[Now ready.]

---

ALDER SMITH, M.B. LOND., F.R.C.S.

Resident Medical Officer, Christ's Hospital, London.

**RINGWORM: ITS DIAGNOSIS AND TREATMENT.**  
Third Edition, rewritten and enlarged, with Illustrations,  
fcap. 8vo, 5s. 6d.

---

FRANCIS W. SMITH, M.B., B.S.

**THE SALINE WATERS OF LEAMINGTON:** Chemi-  
cally, Therapeutically, and Clinically Considered; with  
Observations on the Climate of Leamington. Second Edition,  
with Illustrations, crown 8vo, 1s. *nett.*

---

C. W. SUCKLING, M.D. LOND., M.R.C.P.

Professor of Materia Medica and Therapeutics at the Queen's College,  
Physician to the Queen's Hospital, Birmingham, etc.

**ON THE DIAGNOSIS OF DISEASES OF THE  
BRAIN, SPINAL CORD, AND NERVES.** With Illus-  
trations, crown 8vo, 8s. 6d. [*Just published.*]

---

JOHN BLAND SUTTON, F.R.C.S.

Lecturer on Comparative Anatomy, Senior Demonstrator of Anatomy, and  
Assistant Surgeon to the Middlesex Hospital; Erasmus Wilson  
Lecturer, Royal College of Surgeons, England.

**LIGAMENTS: THEIR NATURE AND MORPHO-  
LOGY.** Wood engravings, post 8vo, 4s. 6d.

---

FREDERICK TREVES, F.R.C.S.

Hunterian Professor at the Royal College of Surgeons of England; Surgeon  
to, and Lecturer on Anatomy at, the London Hospital.

**THE ANATOMY OF THE INTESTINAL CANAL  
AND PERITONEUM IN MAN.** Hunterian Lectures,  
1885. 4to, 2s. 6d.



JOHN R. WARDELL, M.D., F.R.C.P.

Late Consulting Physician to Tunbridge Wells General Hospital.

**CONTRIBUTIONS TO PATHOLOGY AND THE  
PRACTICE OF MEDICINE.** Medium 8vo, 21s.

---

FRANCIS WELCH, F.R.C.S.

Surgeon-Major, A.M.D.

**ENTERIC FEVER:** its Prevalence and Modifications;  
Etiology; Pathology and Treatment; as illustrated by  
Army Data at Home and Abroad. Demy 8vo, 5s. 6d.

---

DAVID YOUNG, M.C., M.B., M.D.

Licentiate of the Royal College of Physicians, Edinburgh; Licentiate of the  
Royal College of Surgeons, Edinburgh; Fellow of, and late  
Examiner in Midwifery to, the University  
of Bombay, etc.

**ROME IN WINTER AND THE TUSCAN HILLS  
IN SUMMER.** A Contribution to the Climate of Italy.  
Small 8vo, 6s.

---

HERMANN VON ZEISSL, M.D.

Late Professor at the Imperial Royal University of Vienna.

**OUTLINES OF THE PATHOLOGY AND TREAT-  
MENT OF SYPHILIS AND ALLIED VENEREAL  
DISEASES.** Second Edition, Revised by M. von ZEISSL, M.D.,  
Privat-Dozent for Diseases of the Skin and Syphilis at the Im-  
perial Royal University of Vienna. Translated, with Notes, by  
H. RAPHAEL, M.D., Attending Physician for Diseases of Genito-  
Urinary Organs and Syphilis, Bellevue Hospital, Out-Patient  
Department. Large 8vo, 18s. [*Just published.*]

---

**CLINICAL CHARTS FOR TEMPERATURE OBSERVATIONS, ETC.**

Arranged by W. RIGDEN, M.R.C.S. Price 7s. per 100,  
1s. per doz., 15s. per 250, 28s. per 500, 50s. per 1000.

Each Chart is arranged for four weeks, and is ruled at the back for making  
notes of cases; they are convenient in size, and are suitable both for hospital  
and private practice.

---

PERIODICAL WORKS PUBLISHED BY H. K. LEWIS.

*THE NEW SYDENHAM SOCIETY'S PUBLICATIONS.* Annual Subscription, One Guinea.

Report of the Society, with Complete List of Works and other information, gratis on application.

*THE NEW YORK MEDICAL JOURNAL.* A Weekly Review of Medicine. Annual Subscription, One Guinea, post free.

*THE THERAPEUTIC GAZETTE.* A Monthly Journal, devoted to the Science of Pharmacology, and to the introduction of New Therapeutic Agents. Edited by Drs. H. C. Wood and R. M. Smith. Annual Subscription, 10s., post free.

*THE GLASGOW MEDICAL JOURNAL.* Published Monthly. Annual Subscription, 20s., post free. Single numbers, 2s. each.

*LIVERPOOL MEDICO-CHIRURGICAL JOURNAL*, including the Proceedings of the Liverpool Medical Institution. Published twice yearly, 3s. 6d. each number.

*THE INDIAN MEDICAL JOURNAL.* A Journal of Medical and Sanitary Science specially devoted to the Interests of the Medical Services. Annual Subscription, 24s., post free.

*MIDDLESEX HOSPITAL.* Reports of the Medical, Surgical, and Pathological Registrars for 1883 to 1886. Demy 8vo, 2s. 6d. *net* each volume.

*TRANSACTIONS OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA.* Volumes I. to VI., 8vo, 10s. 6d. each.

---

\* \* MR. LEWIS is in constant communication with the leading publishing firms in America and has transactions with them for the sale of his publications in that country. Advantageous arrangements are made in the interests of Authors for the publishing of their works in the United States.

MR. LEWIS's publications can be procured of any Bookseller in any part of the world.

*Complete Catalogue of Publications post free on application.*

---

*Printed by H. K. Lewis, Gower Street, London, W.C.*









